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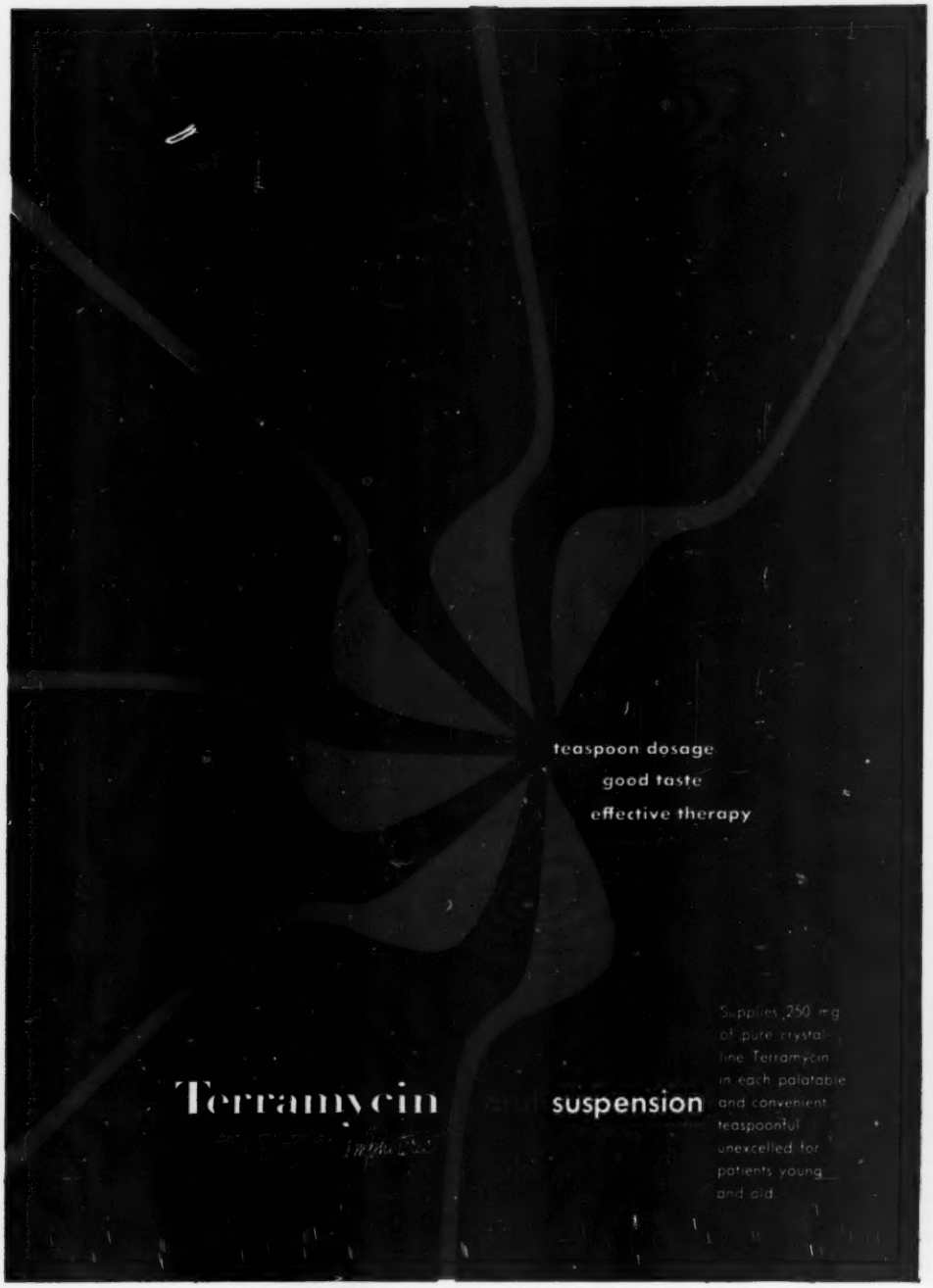
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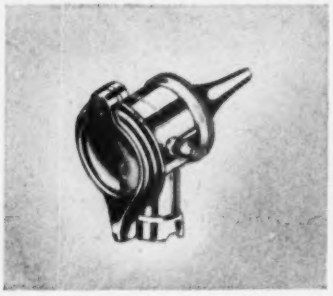

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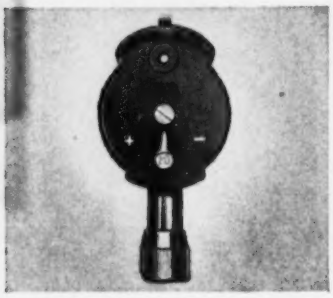

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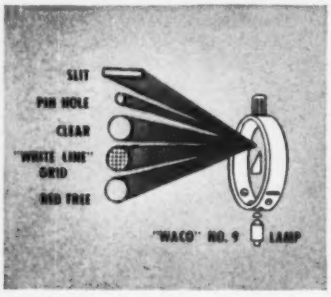

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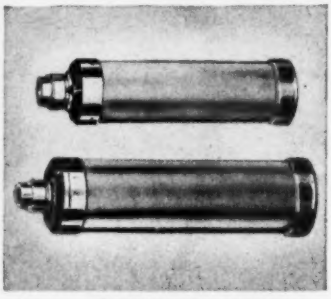

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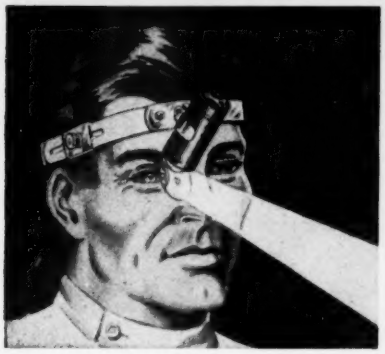

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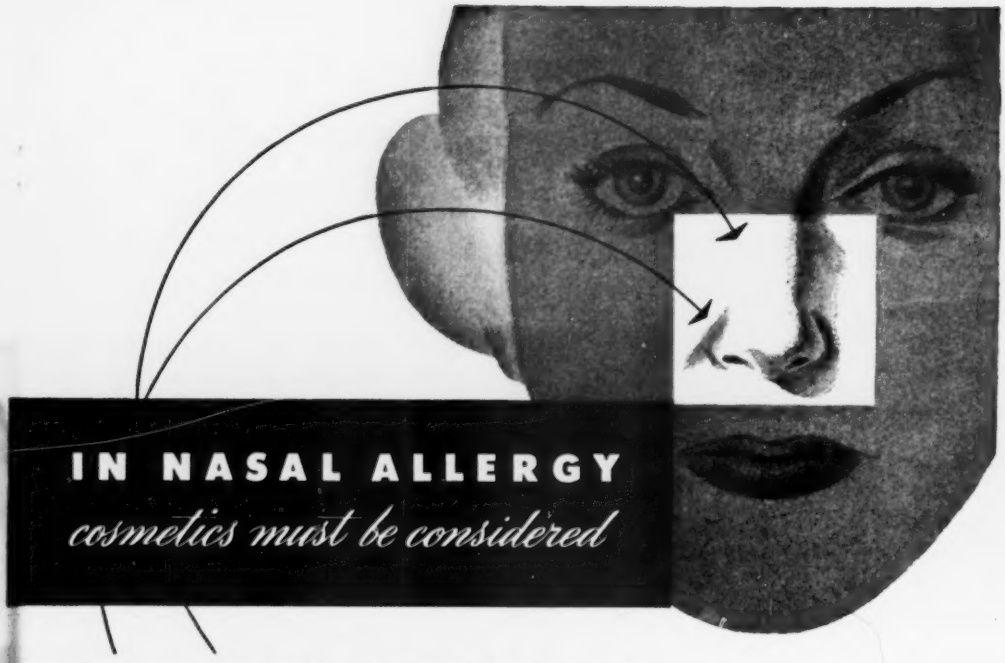

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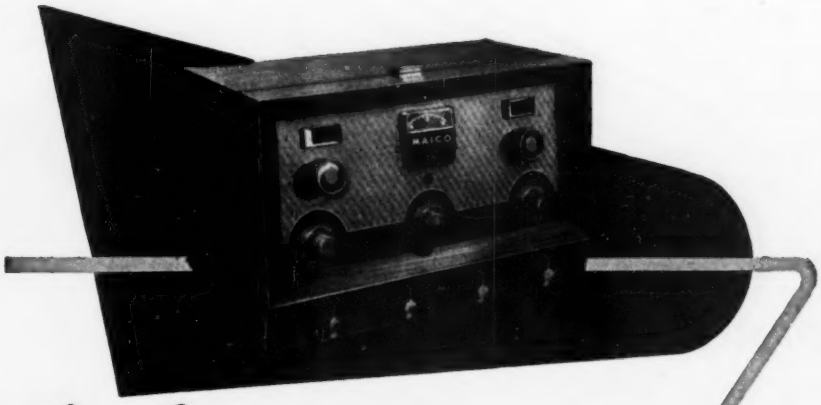

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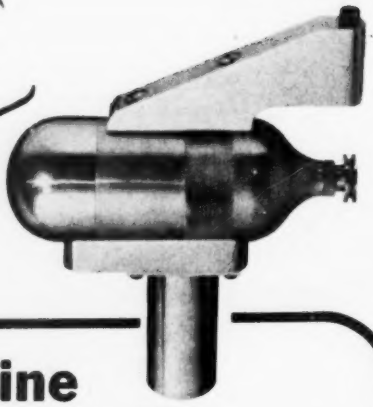

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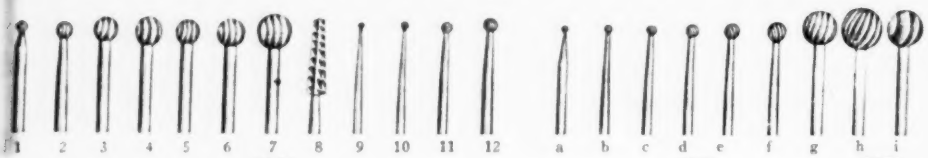

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ANALYSIS OF HEARING LOSS PATTERNS IN A RURAL ILLINOIS SCHOOL SYSTEM

A Study of Ears Receiving Medical Treatment

E. THAYER CURRY, Ph.D.
URBANA, ILL.

GUILD [1] has reported an audiometric analysis of children with reduced hearing acuity who were studied before and after medical treatment continuing over a period of several years. The complete population studied shows, among other things, an increase in hearing acuity at certain frequencies over the six years of the study for some groups of children, regardless of whether or not medical treatment was received. In analysis of the hearing data of the rural Illinois school system under consideration in the present series of studies, the threshold results for boys have been kept separate from those for girls. In the light of Guild's interesting findings, I have considered that an analysis of the present data to discover possible differences by sex in the improvement pattern might prove to be of significance. An effort has been made to break down the cases of the present survey in an attempt to add some insight and explanation to Guild's results.

The present materials cover a group of students who were shown to have a hearing loss at the time of a spring hearing survey. Some of these students subsequently received medical treatment; some did not. All students with hearing losses have been divided into four groups: treated boys, treated girls, untreated boys, and untreated girls. These four groups have been analyzed to determine whether there exist statistically significant differences in amount of shift of the hearing threshold between spring and fall audiometric examinations.

PROCEDURE

The population with which this present analysis is concerned consisted of all the fifth, seventh, and ninth grade children [2] in the Dewitt-Piatt County Health Unit, covering two Central Illinois counties. The public health physician regarded the 6,062 children of this Health Unit as a rural population. Any findings should be considered in the light of the nonurban nature of the population; they may be compared to previous studies dealing with groups regarded as urban and the differences noted.

All the students of the analysis were first given a pure frequency test (15 db. intensity) at seven test frequencies (256 to 11584). Each child who did not pass this screening test was

From the University of Illinois.

1. Guild, S. R.: Nasopharyngeal Irradiation and Hearing Acuity: A Follow-up Study of Children, Laryngoscope **60**:55-76, 1950.

2. These children are of the same general age level as those studied by Guild. He reports an age range of 8 to 13 years at the onset of his study. Guild's study covered a period of about six years—the present study only some six months—but at the outset the two studies deal with individuals of the same age range.

then given an individual pure frequency air conduction audiometric test covering the same seven test tones. As a result of this precise threshold examination, any child who had a threshold raised 30 db. or more at any one or more of the frequencies tested was considered to have a hearing loss and was set aside for special analysis and further study.

The present analysis has concerned itself with the test-retest results on the children shown to have a hearing loss of the extent indicated at the time of the original spring test. The parents of all these children shown to have a 30 db. or greater loss were urged to take their children to their individual family physician for immediate medical examination. After the audiometric survey, the school nurses of the Public Health Unit made individual visits to the homes of the children involved and explained fully the importance of early medical advice and treatment.

As might be expected, not all parents took their children to their family physician for examination. This report is a statistical study of the differences between the groups of treated and of untreated children as found in this particular rural population. Those children with an indicated hearing loss in the spring (March, 1949) were retested in the fall of the same year (October-November). The intervening time gave an opportunity for any medical treatment, if administered, to take effect. Within the time limits of the survey, this was as long a time as could reasonably be allowed for the effects of such treatment to be reflected in the audiometric threshold.

The measured thresholds of the spring and fall tests have been compared by an analysis of variance technique. Thresholds have been thus contrasted and conclusions stated if seemingly justified statistically.

It should be stated here that this study has grown out of what was originally set up as a rather simple, if intensive, hearing survey. As such was the approach, no experimental design was envisioned to measure statistically the changes in hearing thresholds. Because of the introductory and general survey nature of the whole study, it has not been deemed advisable to apply too rigorous statistical techniques to the data collected. At one point an analysis of covariance technique seemed indicated to really establish the validity of the conclusions. This was not carried out in the light of the exploratory nature of the study. Therefore any conclusions not statistically verified should be regarded in the absence of complete statistical treatment as simply general observations.

RESULTS

Three particular results will be considered for the four groups of children treated in this study: girls receiving medical treatment, girls not receiving medical treatment, boys treated, boys untreated.

1. All four of these groups showed improvement in threshold between spring and fall. Tables 1 and 2 display the results of the analysis for the population under study. It should be noted here that the basis of the present study is the threshold value (in decibels) for each specific frequency (or frequencies) which had caused the child to be included in the "hearing loss" category originally. The threshold value for the spring test was compared with the threshold value of the fall retest. Audiograms on all seven frequencies were taken both spring and fall, but for this study only the values for frequencies with 30 db. or greater loss were included. This analysis and the results tabulated in Tables 1 and 2 cannot therefore be regarded as a general shift of the whole audiogram. It is rather for each specific

frequency a study of average shifts in those threshold values which had caused the child to be included in the hearing loss category.

Inspection of Table 1 shows that for purposes of this study all the children having hearing losses have been divided into the four groups previously named.

TABLE 1.—*Shifts in Auditory Thresholds Between Spring and Fall Audiograms*

		Frequency Values, CPS						
		256	512	1024	2048	4096	8192	Average*
Children Showing Increased Acuity at Time of Fall Test								
Spring Thresholds								
Boys	Untreated	35.0	31.5	33.2	41.8	43.2	47.9	39.6
	Treated	40.3	40.0	41.9	48.0	39.7	43.2	41.1
Girls	Untreated	40.7	33.8	43.9	20.0	38.0	42.7	41.3
	Treated	36.7	40.0	36.9	33.3	40.7	39.0	38.0
Fall Thresholds								
Boys	Untreated	13.4	15.0	17.8	18.6	21.1	29.7	20.9
	Treated	12.8	16.2	18.5	16.0	20.5	15.0	16.7
Girls	Untreated	13.9	14.4	14.3	9.4	13.8	16.7	14.2
	Treated	15.0	17.3	14.4	8.3	11.4	16.0	14.6
Average Increase in Acuity								
Boys	Untreated	21.6	16.5	15.3	23.2	22.0	18.2	18.7
	Treated	27.5	23.8	23.4	27.0	19.2	28.2	24.4
Girls	Untreated	26.8	25.2	29.6	28.7	27.9	26.0	27.1
	Treated	21.7	22.7	22.5	25.0	29.3	23.0	23.4
Children Showing Decreased Acuity at Time of Fall Test								
Spring Thresholds								
Boys	Untreated	30.0	11.7	15.0	47.5	31.9	15.3	21.7
	Treated	22.8	17.8	22.5	10.0	29.3	28.0	24.9
Girls	Untreated	21.3	26.3	17.0	38.1	41.7	62.5	27.9
	Treated	22.0	20.0	33.7	40.0	33.3	15.0	27.1
Fall Thresholds								
Boys	Untreated	41.7	36.7	32.5	57.5	46.2	32.8	38.3
	Treated	41.1	38.6	40.0	30.0	43.6	46.3	42.9
Girls	Untreated	33.8	37.5	35.0	37.5	60.0	77.5	42.3
	Treated	38.0	36.7	50.0	58.3	50.7	43.3	46.4
Average Decrease in Acuity								
Boys	Untreated	11.7	25.0	17.5	10.0	14.4	17.5	16.6
	Treated	18.3	20.8	17.5	20.0	14.3	18.3	18.0
Girls	Untreated	12.5	11.3	18.0	17.5	22.0	15.0	14.4
	Treated	16.0	16.7	16.3	18.3	23.4	28.3	19.3

* Average for the six test frequencies.

TABLE 2.—*Increase and Decrease of Hearing Acuity Among Four Groups of Students with Hearing Loss*

		Children Showing Increased Acuity			Children Showing Decreased Acuity			All Children*	
		N†	%	Average‡ Threshold Shift, Db.	N	%	Average Threshold Shift, Db.	Net Shift§ in Threshold, Db.	(N)
Boys	Untreated	123	77.4	18.7	36	22.6	16.6	11.4	(159)
	Treated	90	65.2	24.4	48	34.8	18.0	9.7	(138)
Girls	Untreated	86	78.2	27.1	24	22.8	14.4	18.0	(110)
	Treated	49	70.0	23.4	21	30.0	19.3	10.6	(70)

* Average of all children in group, increased and decreased.
† N, number of thresholds measured.
‡ Average of six frequencies.
§ Net shifts are all improvements.

In addition, each of the four groups has been further subdivided: first, those children whose fall audiogram was better than the spring test, and second, those children whose fall test was poorer than the spring test. Table 1 includes for each of these groups the spring and fall average audiometric threshold at each test fre-

quency; the extent of the average threshold shift is indicated. These threshold differences have been analyzed by analysis of variance; such an analysis was carried out at each frequency for each of the four treatment categories. These results may be summarized as follows:

(*a*) Treated boys showing improvement had an average threshold shift of 24.4 db.; the average threshold improvement for untreated boys was 18.7 db. This difference is not statistically significant. In other words, both treated and untreated groups of boys contained some children who showed an improvement in threshold; the difference in improvement was not of a magnitude to be of statistical significance.

(*b*) The group of treated girls showing improvement had an average threshold shift of 23.4 db.; the average threshold improvement for untreated girls was 27.1 db. As with the boys of the test, some of the treated and some of the untreated girls showed threshold improvement; the difference in magnitude of shift between these two groups was not statistically significant.

(*c*) Children who showed improvement without medical treatment were next compared to see whether there were differences between the boys and the girls. Untreated girls of this group showed an average threshold shift of 27.1 db. compared to a 18.7 db. shift for the untreated boys. The analysis of variance showed this difference to be statistically significant at the 1% level of confidence.

(*d*) For the treated children showing improvement differences between threshold shifts were small. The shift for treated girls was 23.4 db.; treated boys averaged a shift of 24.4 db. This difference is not statistically significant.

These tabulations can therefore be summarized by saying that some girls and some boys, whether or not receiving medical treatment, tended to show an improvement in threshold. These improvements tended to be of the same magnitude and without statistical differentiation as to either sex or treatment, except that untreated girls tended to improve more than untreated boys. This one significant finding may be related to the fact that boys of the population tended to have a greater loss for high tones than did the girls.[3] Improvement of a greater order might be expected to occur in the low frequencies for girls, and this was shown by the study [3] also.

In statistical consideration of these shifts, each of the four groups has been considered as composed of two subgroups, those who improved and those whose threshold acuity decreased. In order to fully evaluate the significance of the threshold changes involved, it is not really adequate to lump together those who improved and those who showed decreases and then to average out the threshold results. An analysis of covariance is immediately suggested as a statistical technique designed to filter out the mean squares of the two groups. This would reduce the size of the remainder variance and might indicate that more of the differences in threshold shifts are actually of statistical significance. As has been previously pointed out, however, this study is a preliminary investigation in this area, and I considered that my purpose would have been accomplished if the major trends were indicated. Since these trends agree with the more general findings of Guild, I am

3. Curry, E. T.: An Analysis of Hearing Loss Patterns in a Rural Illinois School System: II. Considerations for Specific Frequencies, Am. J. Dis. Child. **80**:254-259, 1950.

content to view the present results in a general way. In the absence of factual dissidence, it is not deemed appropriate at this time to carry out a more rigorous statistical analysis of the type indicated.

2. The proportion of thresholds showing improvement tended to be the same for both sexes, as shown in Table 2. Among the children not receiving treatment, 77.36% of the boys showed improvement; 78.18% of untreated girls showed improvement. Among the treated children, 65.2% of the boys and 70.0% of the girls had better thresholds at the time of the second test. The average net shifts in thresholds for all children within a group are also indicated by this table. I feel that the lumping together of the scores from the two subgroups tends to obscure the more general results.

3. Those children whose audiograms showed improvement at the time of the fall test tended to have a poorer threshold in the spring than those who showed decreased acuity at the time of the fall audiogram. This is clearly shown in tabular form by Table 1. Inspection of this table will show that the great majority of average thresholds (19 of 24 measurements taken at the six test frequencies) among the children who showed decrease were better at the spring test than the comparable frequency thresholds for those children who showed improvement at the fall test. These specific threshold averages have not been studied by an analysis of variance technique, but casual inspection should indicate the high probability that the difference in levels of the two groups at the time of the spring test was statistically significant. This may be summarized in a general nonstatistical manner by reporting that in a population with reduced hearing acuity some thresholds will tend to improve and a smaller number will decrease in acuity. The general practice of averaging these two different groups together may tend to thoroughly confuse the resulting averages as far as interpretation of results is concerned. Real differences in threshold shifts of the two groups may be partially or even completely obscured by such averaging.

Comparison of the threshold values in Table 1 will show that there is no statistical difference between the spring thresholds for those who availed themselves of medical treatment and those who did not. The average thresholds for the groups showing improvement were as follows: untreated boys 39.6 db., and untreated girls 41.3 db.; treated boys 41.1 db., and treated girls 38.0 db. These four values do not represent statistically significant differences among themselves. The spring thresholds for the groups showing decrease are also drawn from the same population. These values are as follows: untreated boys 21.7 db., untreated girls 27.9 db.; treated boys 24.9 db., and treated girls, 27.1 db.

SUMMARY AND CONCLUSIONS

This study has been concerned with changes between spring and fall audiometric thresholds in a group of rural Illinois children of the fifth, seventh, and ninth grades. The measured changes in thresholds in children who received medical treatment, as well as in those who did not receive medical treatment, have been studied by an analysis of variance technique. The nature of the medical treatment was diversified and was in every instance administered by the family physician. In the light of the

previous discussions, the following conclusions seem well justified for the population under test:

1. Thresholds in children who received medical treatment tended to show the same improvement as thresholds in children who did not receive treatment.

2. The proportion of thresholds showing improvement tended to be the same for both sexes.

3. Those children whose thresholds showed improvement at the time of the fall test tended to have a poorer threshold in the spring than did those showing decreased acuity in the fall.

4. Children who show improvement should be studied separately from those whose hearing acuity decreases over the period of a study such as is here reported.

ELECTROLYTES AND NOISE SUSCEPTIBILITY

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THE WORK which has gone into the preparation of this paper has consisted largely of asking certain patients many questions. Their answers have been evaluated to form a theory, which must be controversial, since the material used is subjective in nature.

Few of us will argue with the theory, well supported by histological and clinical evidence, that endolymphatic hydrops, whatever may be its real cause, is the basic pathological factor in Ménière's syndrome. It is therefore not illogical to presume that hydrops may exist in the labyrinth without the classic dizziness of Ménière's syndrome.

Dr. H. L. Williams wrote upon this subject in 1950,[1] under the title "Endolymphatic Hydrops Without Vertigo." In this very good article he points out that endolymphatic hydrops may be considered an extracellular edema involving primarily the scala media of the cochlea. Since it occurred in only 50 out of 1,000 patients in the Mayo Clinic, he considers the condition uncommon. He emphasized certain aspects of the syndrome, as follows:

1. Low-tone hearing loss may predominate, and if the case is a recent one a conduction type of deafness may be simulated.

2. The hearing shows spontaneous fluctuation.

3. In women there may be temporary loss of hearing at the menses. (It is important to note his statement that sodium retention with water imbalance may occur in some women at the menses.)

4. Various allergic phenomena may be present.

In treatment he stresses the use of vasodilators and reports some success in treating the disease in early stages in which the process has not progressed to the stage of degeneration.

In diagnosing this condition, which for want of a better term might be temporarily called "waterlogged cochlea" one is more or less dependent upon history taking and symptoms. On this basis hydrops seems to be a common thing, often showing itself as little more than a sensation of obstruction, fulness, pressure, or numbness in the ear, with minimal hearing loss.

Read at the Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, Salt Lake City, April 30, 1952.

1. Williams, H. L.: Endolymphatic Hydrops Without Vertigo, Tr. Am. Otol. Soc., Inc. **35**:116-142, 1950.

The feeling that the ear is "clogged up" is common to most cases of hydrops with or without vertigo. It often comes on in waves, resembling the attacks of dizziness in Ménière's syndrome. In very early or mild cases it may be the only complaint. It is usually the first symptom to disappear under treatment, and in a way it may serve as an index for this purpose.

Many of the patients questioned admit the use of excess salt in their diet. Some of them have a craving for it and regard the seasoning at least as important as the food itself. An extreme example is cited.

A woman, aged 60, stated that 20 years ago she had had recurrent attacks of whirling dizziness with vomiting. This condition lasted for several years and then gradually disappeared, leaving the right ear totally deaf. Hearing remained fairly good, though it fluctuated in the left ear, which often felt clogged up. During such spells music would sound discordantly metallic, and any sudden noise would be uncomfortably loud. Two days ago she awoke to find most of the useful hearing gone in the left ear, which now felt blocked completely. There was no dizziness, except when she quickly looked up. At mealtimes she used an English salt cellar from which, using a spoon, she covered all food with a generous frosting. Because she craved salt, she used it on toast, in marmalade, and even on ice cream.

She was treated with a low-sodium diet and small doses of potassium salt. In a few days the left ear "opened up," and the hearing returned to become more natural than it had been for years. She quickly lost about 6 lb. (2.7 kg.) of liquid weight.

Her history suggests Ménière's syndrome on the right side 20 years ago and since then a waterlogged ear on the left side. It would seem that the labyrinths took their share of a chronic waterlogging due to excess sodium.

Though this case is extreme, it is surprising how commonly one encounters salt addiction by inquiring about it.

In cases of inner ear deafness, particularly in those with flat or curved-up audiograms, it seems worth while asking the patients the following questions:

1. Is there a definite and clear-cut variation in your hearing from day to day? In other words, do you have deaf days and clear days? (I have observed spontaneous fluctuation in hearing of as much as 30 db. in two cases.)

2. Do you recurrently feel that your ear is clogged up, as if it were full of wax?

3. Do sounds seem discordant, unnatural, or at times too loud?

4. Do you sometimes have disturbance of balance, especially on sudden movement, such as looking up?

5. Do you think that you use more salt on your food than most people do?

As a result of the responses to this questionnaire, those patients whose histories may suggest hydrops have been treated with a low-sodium diet and small doses of potassium salts for a month. Radically sodium-free diets in use some years ago are capable of upsetting electrolyte balance to the point of causing illness. The same may be said of heavy doses of potassium. In those patients whose sodium intake seems reasonable or in those who fail to respond to the above treatment the use of vasodilators, as suggested by Williams, has achieved results. (The allergic tendencies noted by him are often present.) Failure of either method of treatment discounts the probability of hydrops. Practical improvement in hearing is occasional where hydrops is present, but relief from the auxiliary symptoms, such as clogged-up ear, is common. It is for this reason that I suggest that subclinical Ménière's syndrome is not rare.

Turning now to the subject of noise-induced deafness, we know that anyone exposed to a loud enough noise for a long enough time will lose some of his hearing. We also know that some people may lose as much hearing in a few weeks or months as others exposed to the same noise may lose in many years. Such people are called "noise susceptibles." It is discouraging, for instance, to see a pilot, trained at great expense, grounded at the peak of his career because his ears will not long tolerate the noise of the air craft, or a man whose only skill is sheet-metal work forced to abandon his trade or lose his useful hearing. If we knew the cause of this phenomenon, and if we could do something to prevent it, we might reduce the substantial list of acoustic casualties among industrial and service personnel.

On Vancouver Island the forest provides the main industry. The sound of a saw screeching through timber is a loud high-pitched, repulsive noise, the type known to be most likely to cause noise-induced deafness.[2]

Three years ago a patient appeared, complaining primarily of a feeling of pressure-like obstruction in his ears. His perception hearing loss was much greater than would be expected from exposure to sawmill noise for 14 months. He was told that his ears were unusually vulnerable to noise and that he must leave this work or risk his useful hearing. He returned several months later, stating that he had left his job as advised but that he was no better. His ears still felt closed, everything sounded unnatural, and his balance was occasionally insecure. For some reason or another he was asked about his salt-eating habits. His reply would suggest that food was a mere vehicle for seasoning and that he had a craving for salt. Treatment with salt restriction plus potassium salt administration soon removed the symptoms which most annoyed him, but his hearing, though subjectively improved, showed only a minor audiometric gain.

He lost a few pounds of weight, passed much urine, and felt better than before. The case provoked the thought that a waterlogged ear might be vulnerable to noise. Since then 29 patients showing greater or less degrees of noise susceptibility have been examined with this thought in mind. The use of salt in definite excess of normal amounts features in the history of about 80% of these people, and a hydrops syndrome is often present. It also seems that those whose ears are most vulnerable to noise are usually those who most crave salt. An additional five patients who have been exposed to loud sawmill or shipyard noise for many years and whose hearing loss is minimal use little or no seasoning at all. In this group the audiogram drops more abruptly in the region of 4,000 cps than in the former group.

REPORT OF CASES

The following cases are of some interest:

Case 1.—A man, aged 59, stated that every few days for several years his right ear would close abruptly, as if a curtain was drawn across it, and that this condition would cause deafness and tinnitus until his ear opened up again. During these attacks his balance was slightly insecure. Ten years ago he operated a donkey engine in the woods. At his left the engine's whistle blew every few minutes, causing tinnitus and deafness at the end of each working day. Within a year he had permanently lost his hearing on the left side.

He used much salt in his food and cooked his own meals, which for convenience usually featured salt pork. An audiogram showed useless hearing in the left ear and a flat-line loss of 45

2. Wheeler, D. E.: Noise-Induced Hearing Loss, Arch. Otolaryng. **51**:344-355, 1950.

db. in the right. Treatment with sodium restriction and potassium salt administration raised the hearing in the right ear by 30 db. This improvement could have been spontaneous, but the "curtain effect" and dizziness disappeared in less than a month. He was then given flushing doses of nicotinic acid, and a month later an audiogram of the right ear showed only a 10-db. loss.

In treatment the use of a vasodilator was as effective as salt restriction, if not more so. The hearing improvement was unusual.

Case 2.—A young man complained of intermittent waves of deafness accompanied by a feeling of closure in his ears for a duration of several years. He had had repeated inflation of the Eustachian tubes, and his ears felt so obstructed that he had bought a syringe to wash them out himself. He used, according to his statement, as much salt as his food would hold. An audiogram showed almost normal hearing in the right ear and a flat-line loss of 20 db. in the left. The deafness was a minor complaint. Treatment with salt restriction removed the symptoms which most bothered him, and his ears seemed to open up. He lost much weight and passed large quantities of urine. The audiogram remained unchanged. His occupation as a butcher did not involve noise, but he had sold his motorcycle because a few hours' ride would render him deaf until the next day. A day of duck shooting would do the same thing.

This case illustrates the waterlogged ear, with noise susceptibility as an incident in the history.

Case 3.—An overweight man, aged 40, plays the piano in his own dance band several nights a week. Deafness had increased year by year and was aggravated after each night's work. In addition to clogged ear, tinnitus, and occasional slight dizziness, he complained mostly of distorted hearing. He had often been told that he used too much salt at meals and admitted using too much. In his orchestra the drums, usually located to his immediate right, caused an annoying vibration in that ear. The right ear showed twice the hearing loss found in the left by audiogram. Treatment with salt restriction and potassium salt administration caused a weight loss which noticeably changed his appearance. Hearing improvement was subjectively definite, mainly through loss of distortion, though objectively negligible. His orchestra now sounds in tune, and he has told the saxophone player, whom he had been scolding, that he was himself to blame for its apparent harsh tones.

This case illustrates the common symptom of sound distortion so often found in waterlogged ears. For instance, several sawmill workers have said after treatment that the saw "now at least sounds like a saw."

Case 4.—A man, aged 39, has been accountant in a fish cannery for three years. To the immediate left of his office a noisy winch operates five hours a day. His left ear was useless for the telephone for the last year. Both ears felt full. Hearing improved a little when he had a holiday away from the noise. He used much salt. There was a perception hearing loss of 30 db. in the left ear and 10 db. in the right. Treatment with salt restriction and potassium salt administration raised hearing by 20 db. in the left ear and relieved the associated symptoms.

The hearing improvement in such a case is uncommon because degeneration has usually taken place.

The personnel managers of two large sawmills (B. C. Forest Products, Ltd.) became interested in this problem and selected 65 men who were employed in the noisiest part of the mills to answer a questionnaire. They were surprised to find that 40% admitted being hard of hearing. Of these about 70% admitted using more salt in their food than most people do. In most sawmills tablets of sodium chloride are provided in dispensers or buckets to replace sodium and chlorides lost in sweat. Some of the men take the matter seriously and eat the tablets by the handful. It is possible that this practice may not be good. As quoted from the textbook, "Principles Of Internal Medicine," edited by Prof. T. R. Harrison

of the University of Texas,[3] "excessive intake of sodium chloride by healthy subjects leads to an increase in venous pressure which may be followed by slight edema."

COMMENT

The theme of this paper is the suggestion that latent hydrops renders an ear vulnerable to noise-induced trauma. Though the role of sodium and water metabolism is emphasized here, it is not offered as the only cause of hydrops or as a contradiction of Williams' theory of physical allergy. It is of interest to note that both allergy and sodium metabolism are under the dominating influence of the adrenal cortex.

CONCLUSIONS

Clinical evidence is offered to suggest the following: (1) Endolymphatic hydrops in the form of a waterlogged labyrinth often exists in a latent form; (2) the cochlea often shares in extracellular edema, due to excess sodium intake or possibly to individual instability in the control of electrolyte balance; (3) such a cochlea may cause no symptoms, unless it is exposed to the traumatic influence of loud noise, and (4) this condition may explain, at least in part, the phenomenon of noise susceptibility.

It is hoped that this presentation may stimulate others to subject the theory to clinical and laboratory trial. In this way the theory may be disproved or followed to a practical conclusion.

303-5 Campbell Bldg.

3. Harrison, T. R., and others, Editors: Principles of Internal Medicine, Philadelphia, The Blakiston Company (Division of Doubleday & Company, Inc.), 1950, p. 237.

DISCUSSION OF ACOUSTIC NEURINOMA WITH CASE PRESENTATION

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IT IS THE purpose of this paper to present briefly the underlying neuropathophysiological basis for the signs and symptoms of the acoustic neurinoma. Far too frequently diagnoses are made simply by memory of a set symptom complex and laboratory data without cognizance of the fundamental physiological basis for the diagnosis. Since, in the majority of cases, patients with eighth nerve tumor consult first the otolaryngologist, he should be capable of localizing the lesion and determining the extent of the lesion and the probable operability. The following review of the normal neurophysiology of the vestibular mechanism and associated neuropathways affected by acoustic neurinomas should aid one in understanding more readily the reasoning in making the diagnosis.

The cerebellopontine angle is located on the lateral aspect of the brain stem at the junction of the pons and the medulla and is bounded above by the biventral lobule of the cerebellum. The cranial nerves, 5th to 10th inclusive, are in close relationship to one another and to this angle area.[1] Thus, any tumor, primary or secondary, within this area is classified as an angle tumor. Therefore, the term, "cerebellopontine angle tumor" is merely a descriptive one localizing the lesion. It is not necessarily specifically diagnostic of the type of lesion. Most authorities classify acoustic neurinomas within the category of cerebellopontine angle tumors.

Eight per cent of all intracranial tumors are acoustic neurinomas. This high specificity for the eighth nerve is based upon many disputed factors. Some observers believe that the tumor arises from embryonic glial tissue which extends along with the nerve from the brain stem to a distance just within the internal auditory meatus. Since the vestibular portion of the eighth nerve is the shortest sensory nerve in the body, measuring slightly more than ½ in. (1.3 cm.) in length, one can readily see how such an extension is anatomically possible. Thus, there is a potential rest of surplus glial tissue at the site of the internal auditory meatus. Any irritative factor, be it trauma, infection, or pregnancy, is thought to cause proliferation of this excess

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1. Buchanan, A. R.: Functional Neuro-Anatomy, Philadelphia, Lea & Febiger, 1948.

of glial tissue.[2] Other investigators are of the opinion that eighth nerve tumors are derived from the fibrous sheath of the nerve, the sheath of Schwann, and not from the nerve itself. Therefore, these tumors should be called schwannomas, or neurinomas. In the majority of cases an acoustic neurinoma springs from the vestibular portion of the eighth nerve, but it can in some instances arise from the cochlear division. Henschen, in 1910, was the first to discover that true eighth nerve tumor has its origin within the internal auditory meatus and that it is not, as formerly believed, of central origin, invading the internal auditory meatus secondarily. The tumors may vary from pinhead to orange size. Small tumors of the acoustic nerve may exist without producing any neurological disturbances. Hardy and Crowe found six instances of so-called silent acoustic neurinomas in 250 consecutive routine autopsies. None of these persons had exhibited any clinical symptoms during life.[3]

Microscopically, the neurinoma may be an encapsulated, solid, cystic, or myxomatous mass. The palisading of the elongated cigar-shaped nuclei and the interlacing bundles are diagnostic. When this picture is not observed, one usually finds tissue changes representative of a nonspecific fibroma.[4]

Cushing stated that the tumors occur more commonly in females and during the third and fourth decades of life and that they have a greater predilection for the left side. The neurinomas are remarkably slow-growing tumors, so that the symptoms are not severe enough for the patient to seek medical attention until approximately 3½ years after onset of the pathological progress. Apparently, acoustic neurinomas have a hereditary factor, as shown by Gardner. He studied a family tree of 200 members and found 37 cases of tumor. He maintained that these tumors can be transmitted as a true Mendelian factor.[5]

Although, as we have stated, acoustic neurinomas comprise 8% of all intracranial tumors, other types of lesions may be found within the cerebellopontine angle, producing symptoms very similar to those of an eighth nerve tumor. One may find gliomas, cystic tumors arising from the cerebellum, meningiomas, aneurysms, metastatic lesions, and, more rarely, tumors arising from the seventh, sixth, and fifth cranial nerves.

The first, and foremost, symptoms of acoustic neurinoma are deafness and tinnitus, and, significantly, only later in the disease does one find recurrent attacks of vertigo. Since these neurinomas most commonly spring from the vestibular division of the eighth nerve, one would ask why deafness and tinnitus should precede the vertigo. This question has been explained on the basis that, since the tumor is a slow-growing one, there is permitted time for vestibular compensation, while irritation of the cochlear division immediately presents itself as tinnitus and progressive deafness. The type of deafness is of a special interest. Many observers have

2. Morrison, W. W.: Diseases of the Ear, Nose and Throat, New York, D. Appleton-Century-Crofts Company, Inc., 1948.

3. Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions, published in ARCHIVES OF OTOLARYNGOLOGY 1925, 1926, 1927, 1928, 1929, 1932, 1937, and 1940.

4. Lichtenstein, B. W.: Textbook of Neuropathology, Philadelphia, W. B. Saunders Company, 1949.

5. Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, Williams & Wilkins Company, 1947.

found that one does not necessarily obtain a nerve-type deafness curve but does find, paradoxically, a low-tone loss with persistent acuity for high tones. However, later in the disease one does find an absolute nerve deafness. One author offers a theoretical explanation for the observed phenomenon. The cochlear nerve has two components: a central core of longitudinal fibers, which take origin from the middle turn of the cochlea and which represent for the most part speech frequency and a low-tone range, and, about this central core, spiral fibers, which stem from the basal turn of the cochlea and which represent high tones. With extrinsic pressure on this nerve, the peripherally placed spiral fibers may readjust, owing to their anatomical location, while the central, solid core cannot. Thus early in the course of the disease there is a resultant central-core pressure atrophy, clinically producing low-tone loss with little disruption of the high tones. Later, as the space-taking lesion obliterates the internal auditory meatus, there is complete pressure atrophy, producing an absolute nerve deafness.[5]

Of diagnostic significance are the mass caloric responses, which must be tested for in every case of suspected neurinoma. One finds on calorimetry hypoactivity to complete loss of function of both the horizontal and the vertical canals on the side of the lesion; contralaterally, the vertical canals can be completely inactive, hypoactive, or perverted, while the horizontal canal may show either normal or perverted responses. Classically, there is a dead labyrinth on the diseased side with impairment of the vertical canals on the opposite side. As a rule, the degree of vestibular disturbance parallels the size of the lesion. However, this relation does not always hold true. In order that these phenomena may be understood, a brief review of the essential neuroanatomy of the vestibular mechanism is presented.

The vestibular nerve is comprised of fibers which arise from the cristae ampullares of the three semicircular canals and from the maculae of the saccule and utricle. These fibers converge in the bipolar cells of Scarpa's ganglion and continue on to enter the medulla just below the pons. Upon entering the brain stem, most of these fibers terminate in one or more of the four vestibular nuclei, namely, the superior, or nucleus of Bechterew, the medial, or nucleus of Schwalbe, the lateral, or nucleus of Deiters, and the inferior, or nucleus of the descending tract of the vestibular nerve. At the point of entrance of the nerve into the medulla oblongata, the nerve fibers divide into two main parts before reaching the nuclei. One path consists of fibers from the vertical canals, and the other path stems from fibers of the horizontal canal. The horizontal-canal fibers terminate in the medial and lateral nuclei, and from here two distinct tracts are formed: the vestibulo-ocular tract, carrying nystagmus-producing impulses, which enters the eye-muscle nuclei via the medial longitudinal fasciculus, and the vestibulocerebellar tract, carrying vertigo-inducing impulses, which enters the cerebellum via the restiform body. On the other hand, the vertical-canal fibers, after entering the medulla, ascend to the pons before terminating in the superior nucleus. From here fibers are sent on to the eye-muscle nuclei and the cerebellum by way of the vestibulo-ocular and the vestibulocerebellar tracts, respectively. However, the fibers of the vestibulocerebellar pathway, concerned with vertigo, which originate in the vertical canals, reach the cerebellum through the brachium pontis, while its vestibulo-ocular fibers also ascend through the medial longitudinal fasciculus. It should be noted here that the vertical-canal vestibular fibers are mesial to the horizontal-canal vestibular fibers. Also of

significance is the fact that vertical-canal fibers only are present in the pons, while both horizontal- and vertical-canal fibers are in the medulla. There is a direct vestibulocerebellar pathway without brain-stem connection via the restiform body.[6]

The loss of responses on the side of the lesion is simply due to compression of the vestibular nerve by the tumor mass within the internal auditory meatus. As the lesion extends or causes pressure outward into the angle, the opposite vertical-canal fiber tracts are also impaired or completely disrupted. This condition may be explained on the basis of the more mesially placed vertical-canal fibers in the brain stem, in addition to the fact that the vertical-canal fibers alone are present in the pons. Because of the variation and the pressure of the tumor there may be acute disturbances in the circulation of the labyrinth, and attacks much like Ménière's syndrome may occur. The patient falls to the side of the lesion because of the hypoactive labyrinth of the diseased side. The vertigo is usually rotational and purely labyrinthine in origin.

Spontaneous nystagmus is common and is comprised of a slow coarse component towards the diseased side and a quick fine component to the opposite side. The cause of the coarse slow component is the direct or indirect pressure on the ipsilateral part of the cerebellum, which controls fine coordinated movement.[3]

Owing to the proximity of the seventh nerve, and because this nerve may be almost in one common sheath with the eighth nerve, a peripheral facial paralysis may develop later in the course of the disease. It is not at all uncommon to find complete loss of function of the eighth nerve while the seventh nerve is but slightly, or not at all, affected.[6a] There is a decidedly greater inherent resistance and strength of the facial nerve than of the eighth nerve, which has a softer structure.

Should the tumor grow cephalad through the internal auditory meatus, the patient may complain of neuralgic pains or anesthesia referable to the area innervated by the first division of the fifth nerve, loss of corneal reflex, and, if the tumor is large enough, dysfunction of the muscles of mastication on the side of the lesion due to third-division extension. With lateral extension, cerebellar disturbances are represented by adiadokokinesis, dysmetria, and dysergia of both limbs on the side of the lesion. With caudal extension, symptoms referable to the 9th and 10th nerves arise, such as hoarseness, disorders of the heart rate, glossopharyngeal neuralgia, and deep ear pain. With fourth-ventricle compression on medial extension, one finds papilledema and other signs of increased intracranial pressure. With medial growth, the patient may also complain of diplopia, due to compression of the sixth nerve on the side of the lesion. The associated headache is localized either to the occiput or to the forehead. It is important to remember that tumors growing in the internal auditory meatus produce sensory disturbances first and motor manifestations later, while tumors growing toward the meatus give rise to motor findings first and sensory symptoms later. In far-advanced cases, besides the ipsilateral cranial-nerve localizing signs, contralateral motor findings, owing to transmitted cerebellar pressure, may be present. However, contralateral sensory phenomena are rare. It should be borne in mind that the multiplicity of signs can be explained by location, size, or extension of the lesion. Patients with neoplasms in the posterior fossa usually show no evidence of

6. (*a*) Jones, I. H., and Fisher, L.: Equilibrium and Vertigo, Philadelphia, J. B. Lippincott Company, 1918. (*b*) DeJong, R. N.: The Neurologic Examination: Incorporating the Fundamentals of Neuroanatomy and Neurophysiology, New York, Paul B. Hoeber, Inc., 1950.

shock reaction to calorimetric tests. Some patients have a characteristic head tilt, such that the chin points to the side of the lesion and the head is tilted toward the diseased side. This may be due to soreness of the neck muscles with resultant splinting, or it may be explained on a reflex basis, according to some authors.

As the acoustic neurinoma increases in size, there is erosion of the bony circumference of the internal auditory meatus. Skull x-ray examination, especially planigraphy, may reveal a bulbous enlargement of the involved internal auditory meatus. The absence of such roentgenographic findings, however, does in no way rule out the presence of an acoustic neurinoma.

REPORT OF CASE

This case presented some of the classic signs and symptoms of an acoustic neurinoma, in addition to other findings which may easily obscure the diagnosis. Interestingly enough, because of the conflicting signs the patient was not given a thorough neuro-otologic examination, and therefore her condition remained misdiagnosed for four years.

E. F., a 28-year-old white widow, was first seen by us at the ear, nose, and throat outpatient clinic at the Philadelphia General Hospital. Her chief complaints were occipital headache, right-ear deafness, both notably of five months' duration, and ringing in the right ear, of four years' duration.

Her present illness began four years before, when during the first trimester of her first pregnancy, she began to have tinnitus in the right ear. The tinnitus stopped for a while after term, only to return shortly thereafter and continue on to her second pregnancy, two years prior to admission. At that time the tinnitus was of greater intensity and persisted. Five months prior to coming to the clinic she began to have constant throbbing headaches in the right occipital region, which were unrelieved by the usual analgesics. There was an associated constant giddiness. The vertigo varied in intensity, such that rising suddenly to the erect position would result in severe rotational vertigo (the patient was uncertain as to its exact direction) with staggering to the right, necessitating concrete support. During this period she also noted that when she lay on the left ear there was a loss for conversational hearing.

The past history was noncontributory. The family history revealed no deafness or other significant factors.

Physical examination, with a complete ear, nose, and throat survey, showed an essentially normal condition except that both tympanic membranes exhibited marked retraction, the right more than the left. Neurological examination gave normal results for all cranial nerves except the eighth. More specifically, funduscopic examination showed no abnormality, and no facial or corneal anesthesia or paresthesia or evidence of peripheral facial paresis was noted. Station and gait were normal; motor power was normal; the Romberg sign was not elicited; position and vibration senses were normal; deep reflexes were normal, and, finally, the Hoffmann and Babinski reflexes were absent.

Results of clinical and laboratory studies were as follows:

1. Hearing Tests: For the spoken voice there was no hearing in the right ear; Weber's test showed lateralization to the left; the Rinne test was negative on the right; Lombard's test with the noise apparatus gave positive results in the left ear and negative results in the right ear; an audiogram revealed absolute nerve-type deafness.

2. Bárány Testing: Only positive findings are listed. There was no spontaneous nystagmus; turning tests revealed a hypoactive response to the right horizontal semicircular canal. Mass caloric irrigations with water at 68 F. elicited no response from the right labyrinth; stimulation of the left labyrinth with the head 30 degrees forward produced rotary nystagmus to the right, which was delayed and of poor amplitude; with the head back, horizontal nystagmus to the right of good amplitude, with past-pointing to the left, was observed. There was no evidence of shock reaction to testing.

3. Laboratory Tests: Complete blood cell count, urinalysis, and cerebrospinal fluid examination gave normal results, as did the serological studies.

4. Skull Roentgenograms: X-ray studies revealed chronic sclerotic mastoiditis on the right; definite asymmetry of the right internal auditory canal, which was bulbous in outline and at least 1½ times the normal width of its fellow on the opposite side, and a slight degree of erosion along the superior margin of the tip of the right petrous pyramid.

The patient was then transferred to the neurosurgical service, where the diagnosis of right-cerebellopontine-angle tumor was corroborated. A right occipital craniotomy was performed, and an acoustic neurinoma, approximately 2 cm. in diameter, was removed from the right acoustic meatus. The specimen removed was a well-circumscribed, soft tumor mass and was diagnosed pathologically as a perineural fibroblastoma. Within three days after operation there was almost complete absence of the occipital headaches.

SUMMARY

This paper has been an attempt to offer a general basic understanding of cerebellopontine-angle lesions, with special emphasis on acoustic neurinoma. Because of the nebulous symptomatology that may be associated with acoustic neurinomas, technical, accurate vestibular testing as a practical routine office procedure is stressed.

The basic neuropathophysiology within and about the cerebellopontine angle with special reference to acoustic neurinomas is presented.

The case of a woman is presented characterized by an audiogram indicative of a conduction type of hearing loss, loss of low tones, as shown by fork testing, and, lastly, onset of initial symptoms (tinnitus with progressive deafness) with pregnancy. This case does not necessarily fall into the category of classic otosclerosis, but may possibly be a case of acoustic neurinoma, which can give identical symptoms and findings. True, it is not common, but its possible occurrence is certainly worth bearing in mind.

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NONCHROMAFFIN PARAGANGLIOMA (GLOMUS JUGULARE TUMOR) OF MIDDLE EAR

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MORE THAN 100 years ago Valentin [1] described a swelling surrounding the ramus tympanicus of the glossopharyngeal nerve in its course through the tympanic canal which he designated the "gangliolus tympanicus."

In 1878 Krause [2] wrote concerning this swelling around the ramus tympanicus, describing a vascular tissue consisting of "a foundation of connective tissue with elastic fibers; it contains arteries, veins and net-like capillaries. The arteries are branches of the ramulus tympanicus out of the A. pharyngea adscendens, which is 0.12 mm. thick and accompanies the N. tympanicus. The former vascular tissue is characterized by a larger or smaller number of three-sided pyramid-shaped or star-shaped perithelial cells of 0.007-0.015 mm. in diameter, the nuclei of which are about 0.004 mm." Also, "at times these cells can pile up around the twisted vessels in such a way that utricular formations suggest the structure of the G. intercarotica." He suggested the name "glandula tympanica."

Krause's interpretation was not accepted universally, as Watzka,[3] while recognizing a striking peculiarity of the tissue in the tympanic canal, surrounding the tympanic nerve, in the way of cellularity and vascularity, classified it as belonging only to connective tissue and not representing a special organ-like structure. He therefore denied the existence of a glandula tympanica, or paraganglion tympanicum, as it came to be called.[4]

Krause's findings were corroborated and elaborated in a study by Guild,[5] in 1941, which reads as follows:

Read at the Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, Salt Lake City, May 1, 1952.

From the Department of Surgery, Division of Otolaryngology, University of Utah School of Medicine, and Holy Cross Hospital.

1. Valentin, G.: Über eine gangliose Anschwellung in der Jacobsonschen Anastomose des Menschen, Arch. Anat. u. Physiol. **74**:287, 1940.

2. Krause, W.: Die Glandula tympanica des Menschen, Zentralbl. med. Wissensch. **16**: 737, 1878.

3. Watzka, M.: Paraganglion Tympanicum, Anat. Anz. **74**:241 (Aug.) 1932.

4. Mulon, P.: Les Glandes hypertensives ou organes chromaffines, Arch. gén. de méd. **2**:3265, 1904. Szymonowicz, L., and Krause, R.: Lehrbuch der Histologie und der mikroskopischen Anatomie mit besonderer Berücksichtigung des menschlichen Körpers, Ed. 6, Leipzig, Curt Kabitzsch, 1930, p. 448.

5. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man, Anat. Rec. (Supp. 2) **79**:28, 1941.

Human temporal bone sections reveal structures, in several respects like the carotid body, for which the name glomus jugularis is proposed. Usually they are in the adventitia of the dome of the jugular bulb, immediately below the bony floor of the middle ear and near the ramus tympanicus of the glossopharyngeal nerve. Usually there is but a single flattened ovoid glomus, about 0.5 mm. in the longer diameters and about 0.25 mm. thick. Occasionally two or more smaller bodies are present; sometimes one or all are in the canal that transmits the ramus tympanicus through the floor of the middle ear (in one case also along the course of this nerve over the promontory).

Each glomus, wherever located, consists of blood vessels of capillary or precapillary caliber with numerous epithelioid cells between the vessels. Usually, but not always, the vessels are the more prominent feature. Innervation and blood come from the same trunks that supply the carotid body, namely, glossopharyngeal nerve and ascending pharyngeal artery (through its inferior tympanic branch).

The interest of both otologists and histologists in vascular tumors of the middle ear was reawakened when, in 1945, Rosenwasser [6] published the first reported case of a carotid body-like tumor of the middle ear and suggested that it may have developed from the glomus jugulare of Guild.

In 1947, Kipkie [7] reported the simultaneous presence of a chromaffin tumor in the middle ear and a carotid body tumor of the opposite side, so that it is unlikely that they were related. LeCompte, Sommers, and Lathrop [8] added another case, and, in 1948, Winship, Klopp, and Jenkins [9] added two more, in one of which metastasis had occurred to a cervical node, and established six others by reviewing the tissues of tumors previously reported as endotheliomas or hemangioendotheliomas. In 1949, Lattes and Waltner [10] reported eight new cases, in one of which there was metastasis to the liver. The list was expanded by various other authors [11] until, in 1951, Winship and Louzan [12] reported a case and were able to gather 41 other proved cases from the literature. In this critical review other probable cases were excluded for lack of satisfactory evidence or tissue for study.

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6. Rosenwasser, H.: Carotid Body Tumor of Middle Ear and Mastoid, Arch. Otolaryng. **41**:64 (Jan.) 1945.

7. Kipkie, G. F.: Simultaneous Chromaffin Tumors of Carotid Body and Glomus Jugularis, Arch. Path. **44**:113 (Aug.) 1947.

8. LeCompte, P. M.; Sommers, S. C., and Lathrop, F. D.: Tumor of Carotid Body Type Arising in Middle Ear, Arch. Path. **44**:78 (July) 1947.

9. Winship, T.; Klopp, C. T., and Jenkins, W. H.: Glomus-Jugulare Tumors, Cancer **1**:441 (Sept.) 1948.

10. Lattes, R., and Waltner, J. G.: Nonchromaffin Paraganglioma of Middle Ear (Carotid-Body-like Tumor: Glomus-Jugulare Tumor), Cancer **2**:447 (May) 1949.

11. (*a*) Fuller, R. H.: Tumor of the Glomus Jugulare: Report of a Case, Naval Med. Bull. **49**:1141, 1950. (*b*) Lundgren, N.: Tympanic Body Tumors in Middle Ear: Tumors of Carotid Body Type, Acta oto-laryng. **37**:367, 1949. (*c*) DeLisa, D. A.: Tumor of Glomus Jugularis, Arch. Otolaryng. **51**:924 (June) 1950. (*d*) Berg, N. O.: Tumors Arising from Tympanic Gland (Glomus Jugularis) and Their Differential Diagnosis, Acta Path. et microbiol. scandinav. **27**:194, 1950. (*e*) Dockerty, M. B.; Love, J. G., and Patton, M. M.: Nonchromaffin Paraganglioma of the Middle Ear: Report of a Case in Which the Clinical Aspects Were Those of a Brain Tumor, Proc. Staff Meet., Mayo Clin. **26**:25 (Jan. 17) 1951. (*f*) Lewis, J. S., and Grant, R. N.: Nonchromaffin Paraganglioma of the Middle Ear (Glomus Jugulare Tumor), A. M. A. Arch. Otolaryng. **53**:406 (April) 1951. (*g*) Poppen, J. L., and Riemenschneider, P. A.: Tumor of Carotid Body Type Presumably Arising from the Glomus Jugularis, ibid. **53**:453 (April) 1951.

12. Winship, T., and Louzan, J.: Tumors of the Glomus Jugulare Not Associated with the Jugular Vein, A. M. A. Arch Otolaryng. **54**:378 (Oct.) 1951.

In addition to these, in 1948, Czurda [13] reported three cases, but in two only was tissue examined histologically, and the descriptions of microscopic studies given by Köhlmeier [14] in these two cases places them in the category of paragangliomas. In 1950, Graf [15] reported eight cases, the diagnosis in three cases being made from the history, findings, course, and histological descriptions, as the slides were not available for study. In 1951, Weille and Lane [16] reported three cases, in one of which the tumor presented itself simultaneously in both the ear and the nasopharynx of the same side; Tamari, McMahon, and Bergendahl [17] reported two cases, in one of which there were metastases to the liver and lungs; two cases were added by Mattick and Mattick,[18] and one was added by Brown.[19] In 1952 Mattick and Burke [20] reported another, bringing the total with the case here reported to 62.

INCIDENCE

These tumors have been recognized but a relatively short time and have been considered uncommon, but they are being recognized more frequently. The first symptom may appear at any age: In Lundgren's series ages ranged from 22 to 69 years; in the cases collected by Winship and Louzan the average age at the time the diagnosis was established was 43.8 years. In 80% of the cases the patients were women. There is a definite familial tendency.[21]

SYMPTOMS

Subjective symptoms are nonspecific and in common with inflammatory conditions of the middle ear, namely, impaired hearing, tinnitus, dizziness, and headache. When the patient is first seen, there is likely to be aural discharge, a polypoid mass in the external auditory canal, and even facial paresis. Characteristic and suggestive are the following [10]: (1) extreme tendency for the mass to bleed with the slightest trauma; (2) subjective sense of pounding noise and pulsation (synchronous with the pulse); (3) progressive expansive type of growth (in advanced stages destruction of the temporal bone, extension into the cranial cavity, and involvement of the caudal cranial nerves), and (4) generally long duration of symptoms, frequently associated with recurrences of polypoid masses in the external canal following repeated removals.

13. Czurda, O.: Angiomatöse Tumoren des Schläfenbeines, Monatsschr. Ohrenh. **82**:164 (April) 1948.

14. Köhlmeier, W.: Über Glomusartige Tumoren im bereiche des Ohres, Monatsschr. Ohrenh, **82**:158 (April) 1948.

15. Graf, K.: Angiomatous Tumors of the Ear: Tumors of the Glomus Tympanicus, Pract. oto-rhino-laryng. **12**:129, 1950.

16. Weille, F. L., and Lane, C. S., Jr.: Surgical Problems Involved in the Removal of Glomus-Jugulare Tumors, Laryngoscope **61**:448 (May) 1951.

17. Tamari, M. J.; McMahon, R. J., and Bergendahl, E. H.: Carotid Body-like Tumors of the Temporal Bone, with Particular Reference to Glomus-Jugulare Tumors, Ann. Otol. Rhin. & Laryng. **60**:350 (June) 1951.

18. Mattick, W. L., and Mattick, J. W.: Some Experiences in Management of Cancer of Middle Ear and Mastoid, A. M. A. Arch. Otolaryng. **53**:610 (June) 1951.

19. Brown, K. B.: Nonchromaffin Paraganglioma of the Middle Ear, Ann. Otol. Rhin. & Laryng. **60**:692 (Sept.) 1951.

20. Mattick, W. L., and Burke, E. M.: Glomus Jugulare Tumors, Laryngoscope **62**:311 (March) 1952.

21. Bartels, J., cited by Winship and Louzan.[12]

Graf [15] divides the progress of the lesion into four stages: (1) tinnitus (often synchronous with the pulse), impaired hearing, and bulging and redness of the tympanic membrane; (2) tumor mass in the external canal, which bleeds easily; (3) cranial nerve palsies, especially of the 7th, 9th, 10th, 11th, and 12th nerves, seldom of the 5th and 6th nerves, occasionally with destruction of the labyrinth; (4) pressure on the brain.

DIAGNOSIS

When a mass presents in the external canal, the diagnosis is easily made by biopsy. Before a tumor is evident, the drum membrane may have a reddened appearance with bulging in the posteroinferior quadrant, with or without pulsation. The drum membrane may be split and tissue taken from the middle ear for microscopic examination,[22] though incision is likely to provoke bleeding that requires snug packing to control.

Occasionally, in the later stages, the clinical aspects may be those of a brain tumor.[23] If one encounters a jugular foramen syndrome, and particularly if a seventh nerve paresis exists also, the possibility of a paraganglioma should be considered.

In the early stages x-ray studies may not be of diagnostic value, but in the later stages they may contribute essentially to the diagnosis by showing involvement of the jugular foramen or invasion of the petrous pyramid.

TREATMENT

The ideal treatment consists of complete removal of all the tumor if this is possible, for the tumor, though histologically benign, is locally invasive and tends to recur unless radically removed. If it is found attached to the jugular bulb, the bulb may be electrocoagulated [11d] or a section of the bulb removed [24] and the bleeding controlled by packing, though some observers are of the opinion that because of increased mortality risk the bulb should be spared.[16] The operation is not without hazard. Even if the bulb is not involved, hemorrhage may be severe and difficult to control. In one reported case 1,000 cc. of blood was said to have been lost at operation,[8] and in several cases operation had to be done in two stages because of profuse bleeding.[10] In one case after removal of the tumor of the middle ear profuse hemorrhage, presumably from the jugular bulb, was controlled with packing, but bleeding continued into the nasopharynx, requiring a postnasal pack for eight hours.[11f]

It has been the general opinion that radiation is ineffective.[25] On the other hand, Graf [15] states that, in contrast to carotid body tumors, these tumors respond well to radiation. The dosage is recorded in two of his cases: One patient received 7,800 r (9,550 r_0); the other, 3,600 r (4,680 r_0). Its use may be reserved for patients with inoperable conditions or when complete removal cannot be accomplished.

22. Lattes and Waltner.[10] Brown.[19]

23. Dockerty, Love, and Patton.[11e] Graf.[15]

24. Winship, Klopp, and Jenkins.[9] Lundgren.[11b]

25. Winship, Klopp, and Jenkins.[9] Lundgren.[11b] Lewis and Grant.[11f] Weille and Lane.[16] Mattick and Mattick.[18]

PROGNOSIS

Metastases from glomus jugulare tumors are as rare as from carotid body tumors,[9] and in but three cases have metastases been recorded: one to a cervical node,[9] one to the liver,[10] and one to the liver and lungs.[17]

These tumors are uniformly slow-growing (except in children)[15]; yet the prognosis is not good, for demanding symptoms do not develop, as a rule, until complete removal is impossible. In the series of cases collected by Winship and

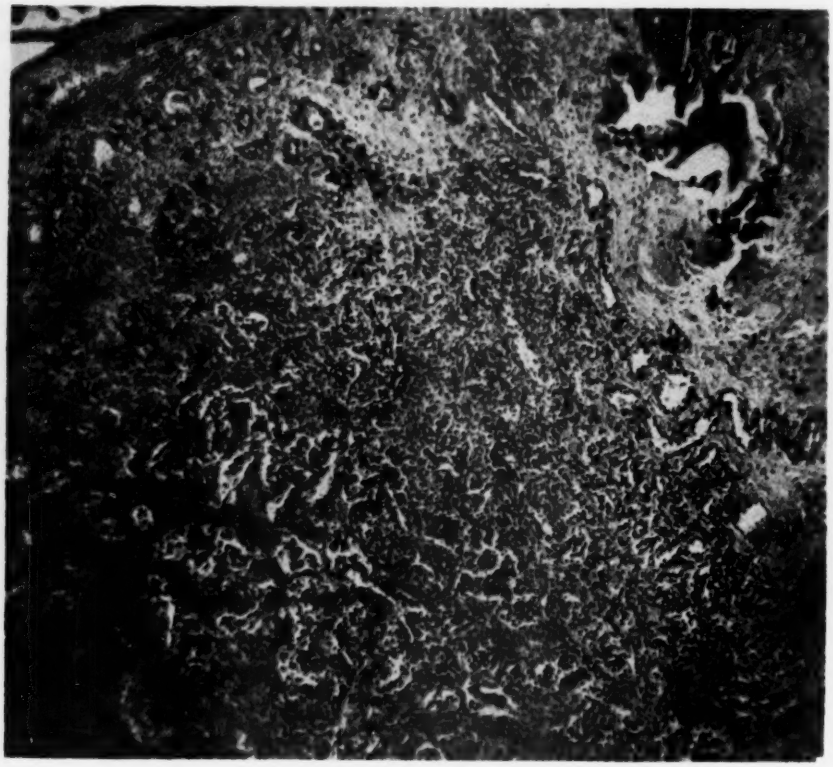

Fig. 1 (W. E. G.).—Nonchromaffin paraganglioma; low power.

Louzan,[12] of the 35 patients who were followed for an average of four years, 12 died either directly or indirectly of their disease, giving a mortality rate of 34%.

Because of the extremely slow growth of the tumor there may be a long interval before symptoms of recurrence are noted. The operative site should therefore be inspected repeatedly at intervals for long periods, so that any recurrence may be dealt with at once. Follow-up examination should include x-ray of the petrous bone.[11b]

REPORT OF A CASE

W. E. G., a man 37 years of age, was first seen on Dec. 8, 1950, complaining of pain in the right ear and headache. When he was 18 years old, the right ear "abscessed," and drainage persisted thereafter. About two years later spontaneous bleeding occurred four or five times

a month, usually at night when he was lying upon the right ear, or after sneezing. Later a growth, which was likened to a sponge, was noted in the ear canal. About 10 years prior to consultation he was seen by an aurist, who attempted to remove a "granuloma" from the external canal but was unsuccessful because of excessive bleeding. Fulguration was then used on several occasions, after which vertigo developed, and a radical mastoidectomy was advised. He was operated upon on Dec. 26, 1940, but the operation was terminated because of severe bleeding. Since then he has had very little trouble, except for occasional head pains, which have become more distressing during the past six months. Two weeks ago a thin, odorous discharge from the right ear developed, and shortly thereafter pain appeared, growing progressively worse.

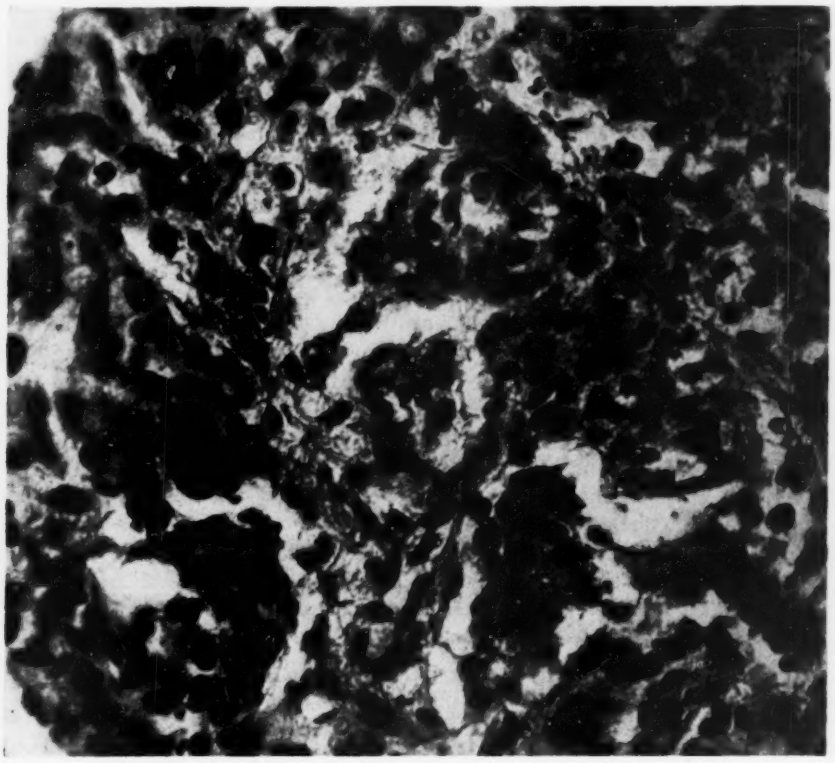

Fig. 2 (W. E. G.).—Nonchromaffin paraganglioma; high power.

Examination showed nothing unusual except for the right ear, which protruded. There was a postauricular scar, and the surrounding area was swollen, red, and tender. Fetid secretion was at the external meatus; the external canal was occluded by a mass having the appearance of a fibrous polyp. There was a conductive-type hearing loss of 66 db. in this ear. There was no nystagmus, past-pointing, or Romberg sign. The temperature was 99.4 F., pulse rate 80 per minute, and blood pressure 140/80. The white blood cell count was 14,800 per cubic millimeter, 68% neutrophiles, 30% lymphocytes, 2% stab forms, the hematocrit reading was 48, and the Kahn reaction was negative. X-ray examination revealed clouding of the right mastoid.

On the following day a radical mastoidectomy was performed using the endaural technique. On exposure of the mastoid frank pus welled up out of the cavity at the site of previous operation. Immediately thereafter bleeding became so profuse that suction would not keep the field clear. The suction tip caught and removed a ball of cholesteatomatous material, and then

a friable, purplish-red mass was seen to be responsible for the bleeding. This was checked with gauze pressure over the mass while the mastoidectomy was revised, and the tumor tissue was then removed piecemeal, pressure being used alternately as bone work progressed until the middle ear was adequately exposed. The tumor filled the antrum, aditus, and middle ear. It was removed completely except from the hypotympanum and around the stapes, and in these places the remaining vestiges were destroyed by electrocoagulation. The bony floor of the hypotympanum was intact. Oxidized cellulose (oxycel®) was laid into the middle ear, and the cavity was packed with petrolatum (vaseline®) gauze. Because of the free bleeding, 500 cc. of blood was given during the operation. A culture from the mastoid yielded a hemolytic Staphylococcus. The postoperative course was uneventful; the cavity healed well and remains dry, and to date there is no evidence of recurrence.

Pathologic Report (Dr. Crichton McNeil).—The specimen consists of eight fragments of tissue, some of which show a white-gray epithelial surface. Other fragments are distinctly hemorrhagic in appearance, resembling a mass of granulation tissue. Histologically the majority of the fragments consist of an angiomatous mass, some of which contain dilated capillaries engorged with red blood cells and others a more highly cellular tumor-like architecture. The tumor masses are apparently made up of endotheliomatous cells in medullary or alveolar patterns, with a generous blood supply always nearby. The cells which make up these columns usually have a small, darkly staining nucleus, which is round with a somewhat abundant cytoplasmic structure and which seems to contain a light-pink-staining material or sometimes is completely vacuolated. Very few mitotic figures are encountered in this mass, so that it is not considered, from the histologic standpoint, to be a malignant tumor. It is considered best that this be identified as a separate tumor of the middle ear, clearly fitting into the group of paragangliomatous tumors described by Lattes [26] and others.

SUMMARY AND CONCLUSIONS

From our descriptions there can be little doubt that the glandula tympanica of Krause and the glomus jugulare of Guild refer to the same structure.

The literature on nonchromaffin paragangliomas (glomus jugulare tumors) of the middle ear is briefly reviewed, 61 cases are collected, and the symptoms, diagnosis, treatment, and prognosis are discussed.

A case of nonchromaffin paraganglioma (glomus jugulare tumor) of the middle ear, having several interesting features is presented. The patient having been operated upon 10 years previously because of a "granuloma" in the external auditory canal, and the operation having been terminated because of severe bleeding, it is reasonably certain that the tumor was present at that time, though no histological examination was made. The present tumor represented, then, not a recurrence but a very slow progression into the mastoid. The 10-year interval was relatively symptomless until the picture of an acute inflammatory process developed. In addition to the vascular tumor, empyema and a cholesteatoma were present. The tumor had no macroscopic connection with the jugular bulb.

26. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid Body, and Aortic-Arch Bodies, Cancer **3**:667 (July) 1950.

CLINICAL EVALUATION OF TRACHEOTOMY IN RESPIRATORY COMPLICATIONS OF POLIOMYELITIS

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WITH THE development of the tank respirator by Drinker,[1] in 1929, the problem of management in the spinal form of poliomyelitis with respiratory insufficiency was so improved that many patients lived and often were rehabilitated to a useful life. The value of this apparatus cannot be overemphasized, but its usefulness in treating patients with the bulbar and bulbospinal forms of the disease was questioned. Wilson,[2] in 1931, felt that it had little, if any, benefit in the treatment or management of these forms of the disease and that it might increase the hazard. In 1932, Wesselhoeft and Smith [3] used the respirator in 30 cases and observed that it had little or no value in the bulbospinal and bulbar cases.

It has been recognized that a great many of the patients with the bulbar form, if they recovered, had little or no residual paralysis. Anderson [4] has shown some very striking figures to substantiate this fact. Death from this form has been universally observed as nearly always being a result of pulmonary complications. Even in 1929, Durand [5] recognized that many patients with the bulbar form died of bronchopneumonia or drowned from aspiration of their own secretions. He reported two successful cases in which the patients were treated by postural drainage but does not take credit for the original idea. Nevertheless, he is believed to be the first to report this treatment in the literature. Smith and Fineberg [6] recognized the value of postural drainage in preventing lung complications or pulmonary insufficiency and, in 1934, recorded improved results in the 1931 and 1933 epidemics in Brooklyn.

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1. Drinker, P., and McKhann, C. F.: Use of New Apparatus for Prolonged Administration of Artificial Respiration: Fatal Case of Poliomyelitis, J. A. M. A. **92**:1658-1660 (May 18) 1929.

2. Wilson, J. L.: Types of Respiratory Failure in Poliomyelitis: Indications for Use of Drinker Respirator, New England J. Med. **205**:597-598 (Sept. 17) 1931.

3. Wesselhoeft, C., and Smith, E. C.: Results of Use of Drinker Respirator in 30 Cases of Respiratory Failure in Poliomyelitis, New England J. Med. **207**:559-563 (Sept. 29) 1932.

4. Anderson, J. A.: Infantile Paralysis in the State of Utah in 1945, Rocky Mountain Conference on Infantile Paralysis, Dec. 16-17, 1946, Denver, Colo., University of Colorado School of Medicine and Hospitals, pp. 130-143.

5. Durand, G. T.: Postural Treatment in Bulbar Infantile Paralysis, J. A. M. A. **93**:1044-1045 (Oct. 5) 1929.

6. Smith, E., and Fineberg, H. I.: Bulbar Poliomyelitis and Its Treatment, J. Pediat. **4**:590-594 (May) 1934.

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As far back as 1931, Wilson [2] suggested that there was a place for tracheotomy in the management of the bulbar or bulbospinal forms of the disease. He mentioned having used it in several cases and even cited one case in which the patient was placed in a respirator for the relief of the bulbospinal involvement. In 1936, Davison [7] suggested that a tracheotomy may be necessary, but he cited no cases. Again, in 1941, Wilson [8] suggested this: "In very carefully studied instances tracheotomy may be life saving." Up to this time it is possible that tracheotomy may have been employed many times, but it was not reported and apparently received little professional recognition. Galloway,[9] in 1943, reported three cases of tracheotomy with two recoveries and was impressed with the miraculous improvement when the secretions were removed from the tracheobronchial tree. He observed that these cases could very easily have been mistaken for marked encephalitis or respiratory center failure. The use of tracheotomy presumably was given a sustained impetus by the Minnesota group during the 1946 epidemic.

Even today there is still some doubt as to its clinical value. In 1949, Wesselhoeft [10] stated that he had yet to see a case of poliomyelitis in which tracheotomy was required. By far the largest group of investigators of this subject are impressed with its value.[11] There is still some disagreement as to its efficiency in many types, and it was with that problem in mind that this investigation was undertaken. This report, then, may be divided into two parts: an analysis of the reported cases and an analysis of the cases studied by me. The specific problem is this: (1) to investigate the clinical value of tracheotomy in the prevention of pulmonary complications and in the management of pulmonary complications in the severe types of poliomyelitis which often result in pulmonary insufficiency as a direct or indirect result of the disease; (2) to determine its value in reestablishing normal deglutition, and (3) to determine if the complications of tracheotomy are sufficiently common to be of concern. The essential, basic principles of respiration will be

7. Davison, W. C.: Poliomyelitis: A Résumé, Am. J. Dis. Child. **52**:1158-1178 (Nov.) 1936.

8. Wilson, J. L.: Symposium on Poliomyelitis: Use of Respirator, J. A. M. A. **117**:278-279 (July 26) 1941.

9. Galloway, T. C.: Tracheotomy in Bulbar Poliomyelitis, J. A. M. A. **123**:1096-1097 (Dec. 25) 1943.

10. Wesselhoeft, C.: Problems of Tonsillectomy and Tracheotomy in Relation to Poliomyelitis, Laryngoscope **59**:726-730 (July) 1949.

11. (*a*) Baker, A. B.: Neurologic Signs of Bulbar Poliomyelitis, Papers & Disc. Internat. Polio. Conf., 1948, **1**:241-244, 1949. (*b*) Bower, A. G., and Associates: A Concept of Poliomyelitis Based on Observations and Treatment of 6,000 Cases in a Four-Year Period (Concluded), Northwest Med. **49**:261-266 (April) 1950. (*c*) Cummings, G. O., Jr.: Tracheotomy in Bulbar Poliomyelitis, Laryngoscope **61**:668-686 (July) 1951. (*d*) Galloway, T. C., and Siefert, M. H.: Bulbar Poliomyelitis: Favorable Results in Its Treatment as Problem in Respiratory Obstruction, J. A. M. A. **141**:1-8 (Sept. 3) 1949. (*e*) Miller, A. H., and Buck, L. S.: Tracheotomy in Bulbar Poliomyelitis, California Med. **72**:34-36 (Jan.) 1950. (*f*) Priest, R. E.; Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, Ann. Otol. Rhin. & Laryng. **56**:250-263 (June) 1947. (*g*) Sjöberg, A.: Mechanism of Suffocation in Spinobulbar Poliomyelitis and Experiences with Operative Treatment, Arch. Otolaryng. **52**:323-340 (Sept.) 1950. (*h*) Strobel, R. J., and Canfield, N.: Tracheotomy in Poliomyelitis, ibid. **52**:341-350 (Sept.) 1950. (*i*) West, H. E., and Bower, A. G.: Treatment of Poliomyelitis Involving the Respiratory System, Am. J. M. Sc. **217**:252-255 (March) 1949. (*j*) Wilson, J. L.: Outline of Essential Treatment of Bulbar Poliomyelitis, J. South Carolina M. A. **44**:354-357 (Nov.) 1948.

reviewed in order that the problem may be clarified and justified. The basis for tracheotomy in complicated cases is to allow a normal exchange of air in the alveoli of the lung for the absorption of oxygen and the elimination of carbon dioxide.

SOME PATHOLOGIC-ANATOMICAL AND PATHOLOGIC-PHYSIOLOGICAL ASPECTS

Pathology of spinal poliomyelitis has been fairly well established for about 20 years, but the concept of the bulbar form is comparatively new. It would seem applicable that certain pathological facts be reviewed. This review may be divided into the effects of the virus on the nervous system in producing respiratory failure and the secondary effects in the lungs.

Bodian [12] concluded in a pathological study that encephalitic symptoms, such as drowsiness, restlessness, disorientation, and coma, occurred in patients who had the severest inflammatory reaction in the brain stem. Small areas of softening often occurred in these patients, but they showed no unusual involvement of the cortex. The only area of the cerebral cortex which he found appreciably affected was the motor area of the precentral gyrus. Edema as a cause for paralysis was not found in the material examined by him. He further stressed the fact that, as far as pathologists are concerned, all cases of poliomyelitis are encephalitic. In the 24 cases studied there were hardly any patients who did not have lesions in most of the cranial nerve nuclei, as well as in the surrounding reticular formation. Rarely did clinical signs of paralysis exist, except in the muscles of the face, pharynx, and larynx. Apparently the remaining nerve fibers were sufficient to maintain function of the muscles supplied by the oculomotor, motor, trigeminal, and hypoglossal nuclei.

Baker [11a] stated that in 90% of the 183 cases of bulbar poliomyelitis studied clinically there was involvement of the 10th cranial nerve. This finding seems to compare favorably with the pathological findings of Bodian. Because of the complexity of the swallowing mechanism any mild involvement would manifest itself early. He further concluded that it was most unusual to have the pure form of respiratory involvement and that most cases were combined with encephalitic and circulatory involvement in varying degrees.

Grulee [13] also found that 96 of 107 patients with bulbar signs had involvement of the 10th cranial nerve. Fifty-five per cent of these had definite dysphagia.

Characteristic lesions are usually not found outside of the central nervous system. Changes in the lungs, while not due to the direct effect of the virus, were observed in varying degrees and were considered to be secondary. Severe lung edema, considerable congestion, bronchopneumonia, and focal atelectasis were the most constant findings.[14] These were presumably brought about by aspiration of secretions and even of food.

12. Bodian, D.: Poliomyelitis: Pathogenesis of Early Stage; Pathologic Anatomy, Papers & Disc. Internat. Polio. Conf., 1948, **1**:62-84, 1949.

13. Grulee, C. G., Jr., and Panos, T. C.: Epidemic Poliomyelitis in Children: Clinical Study with Special Reference to Symptoms and Management of Bulbar Poliomyelitis, Am. J. Dis. Child. **75**:24-39, 1948.

14. Neubuerger, K. T.: Bulbar Poliomyelitis: Pathologic Aspects, Papers & Disc. Internat. Polio. Conf., 1948, **1**:237-240, 1949.

EFFECTS OF HYPOXIA, ANOXIA, AND CARBON DIOXIDE RETENTION

According to Barcroft, Pater, and Van Slyke,[15] hypoxia or anoxia may be brought about in four different ways and likewise are divided into four different types: (1) anoxic anoxia due to decreased oxygen in the inspired air or inability of the air to reach the alveoli; (2) anemic anoxia, as in anemia or carbon monoxide poisoning; (3) stagnant anoxia in which there is poor delivery of the hemoglobin, as in shock or heart failure, and (4) histotoxic anoxia in which the cells fail to utilize oxygen, as in cyanide poisoning.

In respiratory insufficiency one is principally concerned with the first type, in which there is a definite decrease in transfer of oxygen from the alveoli to the blood. Kubicek and co-authors[16] have shown that if the percentage of saturation of hemoglobin is reduced from the normal 96 to 90% the partial pressure of dissolved oxygen in the plasma falls from 100 to 60 mm. of mercury, meaning a reduction of at least 40% in the force which moves oxygen from the blood plasma to the cell. Cyanosis seldom becomes apparent until the oxygen saturation has been reduced to 80%. In the 1946 poliomyelitis epidemic in Minnesota it was noticed that, if the patient experienced an interval of cyanosis, the prognosis was poor.[17] This may be explained on the basis that with approximately 80% saturation of hemoglobin with oxygen the flow of oxygen from capillary blood plasma to nerve cell may be decreased by as much as 65%. Some of the progressive symptoms which Baker[11a] describes as primarily due to hypoxia, regardless of the cause, are divided into four stages: first stage—restlessness, apprehension, anxiety, and sleeplessness; second stage—florid appearance, increasing pulse rate, blood pressure and respiratory effort, euphoria, and a tendency to speak fewer words in one breath; third stage—dyspnea, cyanosis, and occasional panic reaction; fourth stage—cyanosis, circumoral twitching, failure to answer questions, delirium, coma, and terminal shock.

Experimentally it is believed that the cerebral cortex is very much more sensitive to the lack of oxygen than is the medulla.[18] In acute anoxia the effect on the respiratory and circulatory system is manifested by increased pulse, blood pressure, and respiratory rate. Experimental studies[19] have shown that intermittent periods of anoxia may produce irreversible changes in the cortex but may still be compatible with life. Courville[20] studying death after nitrous oxide anesthesia believes that irreversible changes take place after three to eight minutes.

15. Barcroft; Pater, and Van Slyke, cited by Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, Ed. 4, Baltimore, Williams & Wilkins Company, 1945.

16. Kubicek, W. G., and others: Oxygen Therapy in Poliomyelitis: Tracheotomy Inhalator Incorporating Humidification and Optional Use of Positive Pressure for Oxygen Therapy in Patients with Tracheotomy, Arch. Phys. Med. **29**:217-225 (April) 1948.

17. Kubicek, W. G.; Holt, G. W., and Kottke, F. J.: Physiology of Respiration as Applied to Treatment of Bulbar Poliomyelitis, Arch. Phys. Med. **29**:84-88 (Feb.) 1948.

18. Gellhorn, E.: Medulla Oblongata in Anoxia, Arch. Phys. Med. **29**:88-91 (Feb.) 1948.

19. Morrison, L. R.: Histopathologic Effect of Anoxia on Central Nervous System, Arch. Neurol. & Psychiat. **55**:1-34 (Jan.) 1946. Windle, W. F., and Becker, R. F.: Asphyxia Neonatorum: Experimental Study in Guinea Pig, Am. J. Obst. & Gynec. **45**:183-200 (Feb.) 1943.

20. Courville, C. M.: Asphyxia as a Consequence of Nitrous Oxide Anaesthesia, Medicine **15**:129-245 (May) 1936.

Retention of carbon dioxide in the blood even while there is normal saturation of hemoglobin was described in 1926 by Blalock, Harrison, and Wilson.[21] More recently the importance of carbon dioxide retention in the respiratory types of poliomyelitis with normal oxygen saturation has been stressed.[22] This occurs oftener when oxygen is administered and ventilatory failure has developed, owing to a decrease in vital capacity and tidal air. High levels of carbon dioxide in the blood result from inadequate alveolar ventilation. If this is not corrected, it results in blood acidosis, headache, hypertension,[23] disorientation, and loss of consciousness.[24]

TYPES OF RESPIRATORY INVOLVEMENT

Many of the recent studies of the use of tracheotomy as a means to prevent or treat respiratory complications have been described under the term "bulbar poliomyelitis." The term "bulbar" limits the description to the medulla. Wilson [11j] suggests that the term "respiratory poliomyelitis" would better fit and describe the clinical picture of this symptom complex, and, in 1931,[2] he proposed three types of respiratory failure: (1) by actual paralysis of the primary muscles of respiration; (2) by direct involvement of the respiratory center, and (3) indirectly by pulmonary aspiration, laryngeal spasm or paralysis, and pulmonary edema. Sweet,[25] Galloway,[26] and others [27] accepted this description of the causes for respiratory failure in poliomyelitis.

MECHANISM OF PRODUCTION OF RESPIRATORY COMPLICATIONS AND FAILURE

There are certain fundamental physiological states which must be maintained at all times to keep the respiratory system regulated and to prevent any condition from developing which would interfere with the normal exchange of oxygen and carbon dioxide.

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21. Blalock, A.; Harrison, T. R., and Wilson, C. P.: Partial Tracheal Obstruction: Effects on Circulation and Respiration of Morphinized Dogs, Arch. Surg. **13**:81-95 (July) 1926.

22. (*a*) Bower, A. G.; Bennet, V. R.; Dillon, J. B., and Axelrod, B.: Investigation on Care and Treatment of Poliomyelitis Patients, Ann. West. Med. & Surg. **4**:561-582 (Oct.) 1950. (*b*) Plum, F., and Wolff, H. G.: Observations on Acute Poliomyelitis with Respiratory Insufficiency, J. A. M. A. **146**:442-446 (June 2) 1951.

23. Goldstein, J. D., and Du Bois, E. L.: Effect on the Circulation in Man of Re-Breathing Different Concentrations of Carbon Dioxide, Am. J. Physiol. **81**: 650, 1927.

24. Seevers, M. H.: The Narcotic Properties of Carbon Dioxide, New York J. Med. **44**:597-602 (March 15) 1944.

25. Sweet, L. K.: Acute Anterior Poliomyelitis: Diagnosis, Differential Diagnosis, and Treatment of Bulbar Paralysis and Respiratory Failure, M. Ann. District of Columbia **14**:501-504 (Nov.) 1945.

26. Galloway, T. C.: Management of Respiratory Complications of Poliomyelitis, Arch. Otolaryng. **46**:125-136 (Aug.) 1947.

27. (*a*) Baker.[11a] (*b*) Bower, A. G.; Bennet, V. R.; Dillon, J. B., and Axelrod, B.: Investigation on Care and Treatment of Poliomyelitis Patients: Physiological Studies of Various Treatment Procedures and Mechanical Equipment, Ann. West. Med. & Surg. **4**:686-716 (Nov.) 1950; correction **5**:120 (Feb.) 1951. (*c*) Miller and Buck.[11e] (*d*) Rimer, K. G.: Obstruction of Respiratory Tract in Poliomyelitis and Its Treatment: With Special Consideration of Tracheotomy and Bronchoscopy, Nord. med. **43**:788-790 (May) 1950. (*e*) Sjöberg.[11g] (*f*) Stimson, P. M.: The Diagnosis and Treatment of Bulbar Poliomyelitis, South. M. J. **42**:415-420 (May) 1949. (*g*) Strobel and Canfield.[11b] (*h*) West and Bower.[11i]

When the regulating mechanism in the medulla is diseased, periods of apnea develop. When this occurs, coma can develop even without a paralysis of the muscles of deglutition or larynx and respiratory obstruction may supervene. This possibility has been recognized in other diseases where coma is present and the cough and swallowing reflexes are suppressed.[28] When there is respiratory center involvement, it is usually associated with cranial nerve involvement or marked encephalitic symptoms, and no evaluation can usually be made as an independent cause for respiratory failure.[11a]

Any interference with the function of the 9th to the 12th cranial nerves may produce respiratory obstruction and hypoxia or anoxia, as suggested by Baker,[11a] in one or more of the following ways: (*a*) pooling of secretions due to paretic throat muscles; (*b*) paralysis of the tongue; (*c*) obstruction of the airway due to abductor paralysis of the vocal cords; (*d*) obstruction of the airway due to reflex spasm of the laryngeal musculature; (*e*) accumulations of secretions due to inability to cough, and (*f*) aspiration of vomitus.

To prevent aspiration of secretions in patients with a free airway but with paretic throat muscles, prophylactic measures in the form of postural drainage and continuous or intermittent suction may suffice. Once secretions have been drawn into the trachea, a chain of events often takes place. The more depressed the patient, the less is the ability to remove secretions in the normal way. Strobel and Canfield [11b] have emphasized impairment of the cough reflex in impaired respiration. They consider the commonest causes for this to be paralysis of the vocal cords, paralysis of the abdominal or intercostal muscles or diaphragm, hypoxia, encephalitis, and reduced sensitivity of the laryngeal mucosa.

The use of poorly humidified oxygen by a nasal catheter causing drying of the mucosa and destruction of the cilia in the trachea is recognized as another method of retaining even normal tracheal secretions. Proetz [29] has shown that drying is the most effective way of destroying cilia. Hilding,[30] has shown that there are only certain types of secretions which can be removed by cilia, and he has recognized that insufficiency of the cilia may be a contributing factor as a cause of death in other diseases. When these secretions have been drawn into the trachea and bronchial tree, atelectasis and bronchopneumonia develop. If the minute volume has been reduced sufficiently, anoxia and hypercapnia take place.

Pulmonary edema which may develop without aspiration of secretions materially interferes with the transfer of oxygen from alveoli to capillary. Galloway [31] studying acute laryngotracheobronchitis thought that obstruction of the airway caused a cupping action of the capillaries with secondary congestion, stasis, edema, exuda-

28. Atkins, J. P.: Tracheotomy for Prevention of Pulmonary Complications in Postoperative and Severely Debilitated Patients, J. A. M. A. **146**:241-243 (May 19) 1951.

29. Proetz, A. W.: Essays on the Applied Physiology of the Nose, Ed. 1, St. Louis, Mo., Annals Publishing Company, 1941, p. 239.

30. Hilding, A. C.: Relation of Ciliary Insufficiency to Death from Asthma and Other Respiratory Diseases, Ann. Otol. Rhin. & Laryng. **53**:5-15 (March) 1943; Role of Ciliary Action in Production of Pulmonary Atelectasis, Vacuum in Para-Nasal Sinuses, and in Otitis Media, Tr. Am. Acad. Ophth. **48**:367-378 (July-Aug.) 1944.

31. Galloway, T. C.: Laryngo-Tracheo-Bronchial Obstructions and Their Secondary Effects, Ohio M. J. **36**:851-854 (Aug.) 1940.

tion, and hemorrhage of the alveolar mucosa. Drinker [32] suggests that this may be a result of anoxia which is capable of increasing capillary permeability. There is experimental evidence that long-continued use of 100% oxygen may produce alveolar damage.[33] Another factor which produces pulmonary edema experimentally is a rapid drop in serum albumin.[34] A pronounced drop in serum protein has been shown to be closely related to the clinical severity of poliomyelitis.[11b]

PROBLEM OF TRACHEOTOMY

Until the last few years tracheotomy has been used as a means of preventing asphyxia due to either inflammatory or paralytic disease and to tumors of the larynx. Now its effectiveness is recognized in treating patients who have become debilitated or those in coma where an effective cough is absent.[28] There is little doubt but that this use was stimulated by the almost unprecedented attention that has been given tracheotomy in bulbar poliomyelitis.

Before I continue with this review, something must be mentioned in defense of postural drainage, which is recognized as one of the most efficient ways of preventing asphyxia in those patients who have paralysis of the pharyngeal muscles with the inability to dispose of the salivary secretions. This method has been repeatedly emphasized by those who have had experience with this form of the disease. Galloway [26] has shown by injecting the tracheobronchial tree in healthy chests with contrast media that a Trendelenburg position of 20 degrees is necessary to place the trachea on the horizontal in the average patient and a position of at least 30 degrees is needed for the posterior bronchi to drain by gravity. For years some observers have recognized the effectiveness of placing the patient in the prone position. It has been more recently emphasized by Bourdillon and co-authors,[35] especially in respirator cases. It has the added advantage of allowing the mouth secretions to drain freely on the bed or to be aspirated from the buccal folds as the pharyngeal and tracheal secretions drain toward the mouth. Bower and his associates [27b] studying a small number of respirator cases found that patients in a 15-degree Trendelenburg position after 5 minutes had little drop in the tidal air but after 15 minutes there was an average drop of 20%. In the average case the effectiveness of postural drainage cannot be underestimated or overemphasized.

Nasopharyngeal suction is very effective, for it has the added value that the catheter probably causes less salivation than frequent attempts at oropharyngeal suction. Priest and co-authors [11f] report that, after use of continuous mild suction

32. Drinker, C. K.: Pulmonary Edema and Inflammation: An Analysis of Processes Involved in the Formation and Removal of Pulmonary Transudates and Exudates, Harvard University Monographs in Medicine and Public Health, No. 7, Cambridge, Mass., Harvard University Press, 1945.

33. Paine, J. R.; Keys, A., and Lynn, D.: Manifestations of Oxygen Poisoning in Dogs Confined in Atmospheres of 80 to 100 Percent Oxygen, Am. J. Physiol. **133**:406 (June) 1941.

34. Eaton, R. M.: Pulmonary Edema: Experimental Observations on Dogs Following Acute Peripheral Blood Loss, J. Thoracic Surg. **16**:668-694 (Dec.) 1947.

35. Bourdillon, R. B.; Davies-Jones, E.; Stott, F. D., and Taylor, L. M.: Respiratory Studies in Paralytic Poliomyelitis, Brit. M. J. **2**:539-547 (Sept. 2) 1950.

in some of their early cases, it was abandoned in favor of intermittent strong suction. Bouman and McDonough [36] suggest that a very satisfactory method for determining fairly accurately the length of catheter to be inserted into the nasopharynx is to measure the distance from the external nares to the external auditory meatus less 1½ in. (3.8 cm.) in the adult and ½ to 1 in. (1.2 to 2.5 cm.) in children.

The indications for tracheotomy in treating and preventing pulmonary insufficiency vary somewhat. There are those who believe that it practically never need be done.[10] There are those who cling to the more conventional indications represented by laryngeal obstruction.[37] By far the larger group of investigators are more liberal in their application and suggest tracheotomy as a prophylactic measure before cyanosis has developed.[38] The Los Angeles group [39] are even more liberal and do a great many more prophylactic tracheotomies, as evidenced by the large number done in their series.

Galloway [11d] has pointed out eight indications which in his opinion cover all the possibilities. These are presented because of their completeness, being based on symptomatology and findings: (1) progressive anoxia with secretions in the upper airway; (2) unconsciousness or pronounced restlessness in a patient who does not respond to other treatment in a few minutes; (3) pronounced restlessness or stupor in a patient in a respirator, even if the paralysis is apparently of a spinal type; (4) fluid accumulation not otherwise certainly taken care of in a patient who requires a respirator; (5) bilateral paralysis or spasm of the vocal cords; (6) rapidly progressive bulbar symptoms; (7) grave signs of vasomotor failure, and (8) untrained or inefficient attendants, inadequate equipment, or poor cooperation of the patient, with doubt that the airway will be kept constantly free of secretions.

Kubicek and co-authors [17] believed that the oximeter is very valuable in determining early hypoxia. Others have not mentioned its use, and one investigator [26] does not believe that it is a very definite criterion, because it does not measure the degree of carbon dioxide retention.

Sjöberg [11g] describes the angle symptom, which he believes is an absolute indication for tracheotomy. This, he reports, is due to a paralysis of the muscles which support the hyoid bone and the upper aperture of the larynx. Rimer [27d] also believes that this is a very dangerous sign, and he points out that anoxia often develops when this has become apparent.

There are some who believe that bronchial secretions should be removed by repeated bronchoscopic aspirations in preference to tracheotomy, unless there is obstruction to the airway.[40]

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36. Bouman, H. D., and McDonough, K. B.: Some Modern Aspects of Treatment of Bulbar Poliomyelitis, Wisconsin M. J. **47**:1099-1102 (Nov.) 1948.

37. Stimson.[27f] Sweet.[25]

38. Galloway.[26] Henson, E. B.: Treatment of Acute Bulbar Poliomyelitis, West Virginia M. J. **43**:157-158 (May) 1947. Kubicek and others.[17] Priest and others.[11f] Sjöberg.[11g]

39. Bower.[11b] Miller and Buck.[11e] West and Bower.[11i]

40. (*a*) Lenarsky, M.: Bulbar Poliomyelitis, Arch. Pediat. **67**:250-266 (June) 1950. (*b*) Stimson.[27f] (*c*) Sweet.[25]

Complications arising as a direct result of tracheotomy in other diseases are well recognized, and many investigators [41] reported the incidence and the cause for their development. Priest and co-authors [11f] mention mediastinal emphysema, subcutaneous emphysema, and tracheitis as complications of the tracheotomy but do not mention the number of cases. Miller and Buck [11e] in a reported series of 198 cases studied at the Los Angeles County Hospital report one case of mediastinal emphysema, two cases of pneumothorax, and three cases of troublesome bleeding wounds. The other articles reviewed either did not mention these complications or specifically stated that there were none.

The use of the O'Dwyer intubation tube was considered by Priest and his colleagues [11f] in their reported cases, but actually it was used once and discarded. The anesthetic intubation tube was never employed by them as an airway, except during the procedure of tracheotomy.

While a tracheotomy is being done on patients who require artificial respiration, Peterson and Ward [42] recommend the use of the Monaghan chest respirator. Bower and co-authors [22a] mention the use of the Bennet flow-sensitive pressure breathing unit in their cases which was adapted to poliomyelitis patients. It was found to be more useful in ventilating the lungs either by mask or through a bronchoscope with a special adapter during tracheotomy.

Once a tracheotomy has been done, many problems arise, especially in those cases in which the patients must be placed in a tank respirator. The problem of the respirator collar decreases the effectiveness of the tracheotomy, unless it can be pushed well down below the tube. For this reason the tracheotomy is done high, through the second and third tracheal rings, if possible. As a practical emergency measure for retracting the collar, Allen and Landmesser [43] suggest the use of an abdominal retractor attached to a string and tied inside the respirator. Where preparations have been made, an iron bar is bent in such a fashion that it pushes the anterior part of the collar downward over the chest, so that the tracheotomy tube is well exposed in all but the short-necked person. An air seal for reducing the problem of air leakage around the collar in a tracheotomized patient has been described by Damshki and McClure.[44]

During the period from 1946 through 1949 a great many cases of tracheotomy were studied at the Los Angeles County Hospital, and a number of new devices were developed by Bower and his associates [22a] which they believed helped to solve many of their problems. To prevent having to push the collar plate over the

41. Fitzhugh, G. S.: Tracheotomy: Study of 100 Consecutive Cases, South. M. J. **34**:1116-1121 (Nov.) 1941. Forbes, G. B., and Salmon, G. W.: Mediastinal Emphysema and Pneumothorax Following Tracheotomy: Report of 4 Cases, J. Pediat. **23**:175-183 (Aug.) 1943. Goldberg, J. D.; Mitchell, N., and Angrist, A.: Mediastinal Emphysema and Pneumothorax Following Tracheotomy for Croup, Am. J. Surg. **56**:448-454 (May) 1942. Michels, M. W.: Pneumothorax and Mediastinal Emphysema Complicating Tracheotomy, Arch. Otolaryng. **29**:842-852 (May) 1939. Simpson, W. L.: Pneumothorax Complicating Tracheotomy in Fulminating Laryngotracheobronchitis: Report of a Case, ibid. **26**:411-414 (Oct.) 1937.

42. Peterson, R. L., and Ward, R. C.: Tracheotomy in Poliomyelitis Simplified with New Respirator, Arch. Otolaryng. **48**:156-158 (Aug.) 1948.

43. Allen, T. R., and Landmesser, C. M.: Adjusting the Collar for Tracheotomy in a Patient in a Respirator: A Simple Emergency Method, J. A. M. A. **142**:478 (Feb. 18) 1950.

44. Damshki, A. E., and McClure, E. J.: Air Seal Device for Tracheotomized Respirator Patients, Am. J. Nursing **49**:448-449 (July) 1949.

head each time, it was made into an upper and lower section. The upper section contained a depressed shield so as to push a horseshoe-shaped collar downward over the chest. This collar was so constructed that the arms of the collar overlapped behind the neck, completing the seal. They believed that this device solved their problems in placing a patient in and out of the respirator.

For better oxygenation of the blood Glaser [45] uses oxygen direct into the tracheotomy tube but recommends that the amount used be limited to the estimated volume of tidal air of the patient. Elam and his associates [46] have shown that if suction was continued through the tracheotomy tube during the inspiratory phase oxygenation of the blood was dramatically decreased, as studied with an oximeter. Kubicek and co-authors [16] have reported the use of a tracheotomy inhalator incorporating humidification and optional use of positive pressure in oxygen therapy. Fifty per cent oxygen and helium was used through this inhalator, although for limited periods 100% oxygen was used. It was used for 39 patients, 21 of whom were in the mechanical respirator. While no other reports of cases in which this inhalator was used have been reviewed, it was believed by my associates and me to be of definite value in those cases where pulmonary edema had developed.

The Bennett positive-pressure respirator attachment [22a] was found to be most useful by the Los Angeles group in respirator cases, especially for the patients with pulmonary edema. It was so constructed that positive pressure was applied only during the inspiratory phase of respiration. A humidifier attachment was also constructed which was used to control humidity. A new type of tracheotomy tube was also developed by this group which deviates from the standard type in not being a circular arc. It was so constructed that the inner tube was made of plastic material, so that its flexibility would adapt to the metal outer tube. An oxygen catheter adapter for this tracheotomy tube was also devised. These improved instruments have not been reported in use by others.

Table 1 represents actual tabulated cases taken from the literature and reported as a series or as individual cases. There are 502 cases of tracheotomy, representing nine groups or persons reporting. It need not be emphasized that there are probably hundreds of cases in which tracheotomy has been done and has not been reported. In all those investigators reporting there seemed to be definite belief that it was lifesaving in a great many cases. Lenarsky did not share the same enthusiasm as the others. This table gives some idea as to the great variation in the percentage of tracheotomies done in bulbar cases and the variation in death rate.

Some striking figures have been reported by Bower and his associates [24] in all cases in which the patients were treated from 1946 through 1949. In this large series of cases by doing a greater number of tracheotomies to the point of prophylaxis the total case fatality rate was reduced from 3.8% in 1946 to 1.95% in 1949. The total respiratory case fatality rate was reduced from 79% in 1946 to 17% in 1949. Some of this reduction could be explained by improved management in general.

45. Blaser, D. F.: Tracheotomy in Bulbar Poliomyelitis, J. Pediat. **27**:560-564 (Dec.) 1945.

46. Elam, J. O.; Hemingway, A.; Gullickson, G., and Visscher, M. B.: Impairment of Pulmonary Funciton in Poliomyelitis: Oximetric Studies in Patients with the Spinal and Bulbar Types, Arch. Int. Med. **81**:649-665 (May) 1948.

TABLE 1.—*Summary of Reported Cases, 1943-1950*

Reporting Group or Physician	Total Bulbar or Complicated Cases	Cases Respirator Required	Cases Respirator Not Required	No. of Tracheotomy Cases	Deaths in Tracheotomy Cases	Deaths in Non-tracheotomy Cases	Complications	Comment
Minneapolis group, 1946.....	400	Not specified	Not specified	75	46	Not specified	Pneumothorax emphysema; none causing death	Estimated conservatively lifesaving in 17 cases
Evanston Hospital, Galloway, 1947-1950	63	Not specified	Not specified	25	(———10———)		Not specified	Was lifesaving in many cases
Strobel and Canfield.....	17	10	None	10	5	1	None	Lifesaving in 5 patients who lived; 3 others might have lived if done earlier
Sjöberg	Not specified	Not specified	Not specified	14	2	Not specified	None	Believe lifesaving in all cases
McLorinan	Not specified	1	2	3	1	None	None	Believe lifesaving and should be done early
Rimer	Not specified	2	None	2	None	None	None	Lifesaving in these 2 cases
Los Angeles County Hospital, Bower and West, 1946.....	129	48	81	14	7	31	In 198 cases reviewed, 1 mediastinal emphysema, 2 pneumothorax, 3 troublesome bleeding	Believe that success in half of patients who lived was due to tracheotomy
1947	Not specified	21	Not specified	8	4	10		
1948	600	280	320	230	(———123———)			
1949	Not specified	130	Not specified	105	(———22———)			
(70) cases Syracuse, N. Y., 1950, Cummings	68	Not specified	Not specified	6	2	4	Not specified	Lifesaving in all cases
Willard Parker Hospital, Lenarsky, 1947	8	Not specified	Not specified	2	2	Not specified	Not specified	Was not impressed, as only 3 of the 10 patients recovered
1948	45	Not specified	Not specified	5	2	6		
1949	126	Not specified	Not specified	6	6	33		

Galloway and Elsen [47] also show a mortality rate of 12.8% for the bulbar and bulbospinal cases for the years 1947 to 1950 inclusive, while outside of Chicago for the same period the mortality rate was 38.3%.

Lenarsky [40a] reporting cases for the Willard Parker Hospital for the years 1933, 1935, 1944, and 1949 shows a rather consistent bulbar fatality rate between 24% and 30.9%. In 1948 it dropped to 17.7%, but there were comparatively few bulbar cases, and three of the five tracheotomized patients lived. Sweet [25] reports cases from the Gallinger Municipal Hospital for the years 1938 to 1945 inclusive and shows a bulbar fatality rate of 8.3%, a bulbospinal-respiratory fatality rate of 75%, and a spinal-respiratory-failure fatality rate of 40%. In the latter most of the deaths were late. No mention is made of doing tracheotomy in these cases.

Early in 1950 a poliomyelitis team was organized to assume the responsibility for improving the care and study of poliomyelitis victims in a general hospital designated as a respiratory poliomyelitis center. I was appointed as the consulting otolaryngologist to assist in the direction and management of those patients afflicted with the bulbar or respiratory type in which there were impending lung complications of obstruction to the airway. Since there had been little opportunity for observation of these patients in the past, except in occasional instances, the challenge required considerable review of the literature in order to become familiar with the indications, management, and expected results.

When this study began, I was cognizant of the most complete study the Minneapolis group had made and the observations and original work of Galloway and smaller groups referred to in this paper. The Los Angeles group was reporting large numbers of tracheotomies, in many instances being done as a prophylactic measure. At that time many of the investigations they made had not been published. In no way was it expected that a large series would be collected, but still with evidence of controversy it was hoped that some valuable conclusions could be drawn.

At the time this study began, the ordinary standard equipment and mechanical aids were in adequate supply, consisting of tank- and chest-type respirators, rocking beds, oxygen equipment, bronchoscopes, and suction equipment, both mechanical and water. Much of the specialized equipment developed in some of the centers was not then available.

Indications patterned very much after those described by Galloway and referred to earlier in this paper were the basis on which many of the tracheotomies were done. These do not appreciably differ from the majority recommending tracheotomy.

All the patients who were critically ill were given special nursing, in most instances by well-trained nurses, though at times experienced nurses were not available. The resident staff was in attendance at all times and, except on occasions, was experienced in the management of poliomyelitis. In most instances the otolaryngologist saw all the patients with swallowing difficulty or those with respiratory difficulty who were critically ill. As thorough an examination as possible was carried out, but in some instances those patients in respirators were not completely examined unless a tracheotomy was inevitable, because the mere examination of the pharynx often caused panic reaction.

47. Balloway, T. C., and Elsen, J.: Bulbar Poliomyelitis: A Respiratory Problem, Laryngoscope **61**:548-564 (June) 1951.

On admission all the patients were studied by spinal tap and physical findings and were admitted either to the medical service or to the pediatric service. At the time of admission all patients who had evidence of bulbar or central nervous system symptoms were not fed by mouth. In addition to intravenous fluids, many of the severely ill patients were given blood transfusions when indicated.

Postural drainage at a position of at least 20 to 25 degrees was attempted for every patient with bulbar signs, but it was difficult to maintain for the patients in the respirator. For those not requiring the respirator the prone position was insisted upon. Nasopharyngeal suction by the stationary catheter method was used in many instances, but frequently the catheter would be moved up and down, causing irritation. The mechanical suction machine used intermittently was found best. Often the water suction when used continuously was ineffective because of the low vacuum used. Pharyngeal suction by mouth was discouraged, as it nearly always upset the patient, causing gagging and increased salivation.

TABLE 2.—*Analysis of All Complicated Cases, 1950-1951*

	No. of Severe Cases	Swallowing Difficulty*	Paralysis of Vocal Cords	Marked Central Nervous System Symptoms†	Pulmonary Complications‡	Clinical Cyanosis	Tracheotomy	Deaths in Tracheotomy Cases	Deaths in Nontracheotomy Cases
Bulbar	16	8	0	4	1	1	3	2	1
Bulbospinal without breathing difficulties	6	2	0	0	0	0	1	0	0
Bulbospinal with breathing difficulties not requiring a respirator	1	1	0	1	0	0	1	0	0
Bulbospinal with breathing difficulties requiring a respirator	15	10	2	6	10	11	8	5	7
Spinal requiring a respirator	12	0	0	0	2	3	2	0	0
Total cases: 270	50	21	2	11	13	15	15	7	8

* Denotes inability to dispose of saliva in a normal way in the presence of muscular paralysis.
† Indicates toxicity and coma believed not due to pulmonary anoxia.
‡ Indicates, in the tracheotomy cases, secretions in the tracheobronchial tree, atelectasis, or bronchial pneumonia, and in the nontracheotomy cases, pulmonary complications, either present post mortem or revealed by bronchoscopy.

Oxygen was administered by nasal catheter, being well humidified by passing it through three bottles of water. In the cases of tracheotomy a cone was placed about 1 in. (2.5 cm.) above the opening when oxygen was used.

When tracheotomy became necessary, it was done over a bronchoscope, except in two respirator cases in which the adult patients were not in extreme danger of asphyxiation but had mucus accumulation which was becoming more difficult to control. Oxygen was given by flowing it through the side airway on the bronchoscope. For those who needed artificial respiration while the tracheotomy was being done, the Monaghan chest respirator was used. In one instance a tracheotomy was done while the tank respirator was operating. This was so difficult to do that no further tracheotomies were attempted without the patient being removed from the tank respirator. An anesthetic intubation tube was used in one instance for two days, but an airway was difficult to maintain, and a tracheotomy was done. In addition to the supportive treatment mentioned previously, penicillin was given intramuscularly to the tracheotomized patients.

During 1950 and part of 1951 up to the time of this writing there were 270 patients with poliomyelitis admitted, with a case fatality rate of 5.5%. Of these

there were 50 with disease described as complicated—bulbar, bulbospinal, or spinal—and 28 of the patients had severe breathing difficulty, with a group fatality rate of 30%. Table 2 gives an analysis of these cases, stressing the points I believe are important. If all cases were reported in this way, a much better understanding of the value of tracheotomy might be estimated. There are 15 cases of tracheotomy; 11 of these were done in 1950. The policy of conservatism was followed during this year. In many instances this meant waiting until clear-cut indications were present. Only four of the patients lived. In 1951 up to the present writing four tracheotomies were done, and four patients lived. The indications were basically the same, except that the tracheotomy was done earlier on anticipation and before clinical cyanosis had taken place.

REPORT OF CASES

A brief résumé of the cases of tracheotomy is submitted as a part of the record. Only details which were believed to be important in determining the effectiveness and indications for tracheotomy are given.

CASE 1.—C. W. B., a boy 5 years of age, was admitted on July 18, 1950, with symptoms of poliomyelitis of two weeks' duration. On admission there was a high temperature, of 105 F., and weakness of the lower extremities, but there were no breathing or swallowing difficulties. Rapid signs of an ascending paralysis developed, and 10 hours later slight breathing difficulty developed. Fourteen hours after admission slight cyanosis developed because of intercostal and diaphragm weakness, and he was placed in a respirator. Slight nasal twang developed 16 hours after admission, and there was no paralysis of the pharyngeal muscles but slight paralysis of the soft palate. The condition remained critical, gradually the patient became more depressed, and one hour before tracheotomy the gag reflex disappeared. Tracheotomy was done over a bronchoscope, and there was a great deal of mucus in the tracheobronchial tree. An x-ray of the chest, done shortly before death which occurred 31 hours after admission, indicated no atelectasis or bronchopneumonia. The patient died with a temperature of 107 F.

Comment.—Although no postmortem examination was done, the lungs seemed to be aerating properly. It is believed in retrospection that this patient should have undergone tracheotomy about 10 hours earlier. Marked central involvement is believed to have taken place, but death due to damage by earlier anoxia cannot be excluded.

CASE 2.—I. L. B., a boy 14 months of age, was admitted on July 19, 1950, with symptoms of an illness for three weeks. He began using accessory muscles for respiration about 12 hours before reaching the hospital. Mild cyanosis on admission had supervened; the child was immediately placed in a respirator, and a tracheotomy was done over a bronchoscope within two hours. There was some extremity paralysis, though not marked. Direct laryngoscopy showed a poor gag reflex and normal vocal cord motion with the trachea full of mucus. The color continued to remain poor in spite of oxygen therapy and never became normal. Gradually anoxia became severer, and the patient died 37 hours after admission. Postmortem examination, in addition to central nervous system findings, showed very marked pulmonary congestion which was thought responsible for death. Positive-pressure oxygen therapy was indicated.

CASE 3.—N. S., a boy aged 7 years, was admitted on July 23, 1950, with symptoms of a stiff neck and a fever of five days' duration. The first difficulty in swallowing began several hours after admission. In spite of a 30-degree Trendelenburg position and though there were few or no signs of lower respiratory paralysis, slight cyanosis began to develop 30 hours after admission. Thirty-three hours after admission a tracheotomy was done because of recurrent periods of cyanosis, increased blood pressure, pulse, and labored respiration. At the time of tracheotomy there was no gag reflex and no muscular tone to the lower pharyngeal muscles, and the vocal

cords were in the paramedian position but did not move. There was no fluid in the trachea. Color and general condition improved, but three hours later he became cyanotic because of poor action of respiratory muscles. For six days he remained in a respirator, was then removed temporarily for varying periods of time, and was removed permanently on the ninth day. On the 10th and 11th days bronchoscopic aspirations were required to remove thick secretions which produced cyanosis. Progressive and steady improvement, though slow, occurred, and swallowing began after 47 days. The tracheotomy tube was removed on the 57th hospital day. There was no swallowing difficulty, excellent cough reflex, and practically no residual extremity paralysis on discharge.

Comment.—Tracheotomy should have been done several hours earlier. Two attacks of bronchial obstruction, which almost caused death, were probably due to ineffective aspiration of the trachea and relaxation of the use of the Trendelenburg position. Without tracheotomy this patient would not have recovered.

CASE 4.—F. B., a boy 9 years of age, was admitted to the hospital on Aug. 7, 1950. Symptoms of poliomyelitis began four days before admission to the hospital. The first difficulty in swallowing began a few hours before admission. Until the third hospital day he did not show signs of respiratory difficulty. At this time he was put in a respirator, with special emphasis placed on postural drainage. A direct laryngoscopy and bronchoscopy done while he was in the respirator revealed little secretion in the pharynx, questionable paralysis of the right vocal cord, and paralysis of the upper and lower pharyngeal muscles and the right side of the palate. The following day a laryngeal crow developed, and an immediate tracheotomy was done which showed both cords in the median position. His further course was uneventful, and he was permanently removed from the respirator on the fifth day. He began to swallow well on the 35th hospital day, and on the 40th day the tracheotomy tube was removed. There was little residual paralysis, except slight paralysis of the upper extremities and the right side of the soft palate and middle constrictor muscles.

Comment.—This was a definite case of laryngeal obstruction due to abductor paralysis. Tracheotomy should have been done 24 hours sooner. The procedure was believed to be lifesaving.

CASE 5.—E. K., a man 24 years of age, was admitted to the hospital on July 28, 1950, with symptoms of a sore throat; he had been acutely ill for one week. Symptoms on admission were principally swallowing difficulties, and on examination the left 9th and right and left 10th cranial nerves were involved. Temperature was 105 F., and the patient was extremely toxic. With postural drainage and nasopharyngeal aspiration an adequate airway was maintained until four hours later, when a convulsion developed, necessitating an immediate tracheotomy, and the trachea was found to be full of mucus. He died immediately after the tracheotomy.

Comment.—Although a postmortem examination was not done, the convulsive seizure and aspiration of mucus were probably the immediate cause of death. For such a severely toxic patient with swallowing difficulty a prophylactic tracheotomy might have proved lifesaving had it been done earlier.

CASE 6.—N. C., a man 28 years of age, was admitted on Aug. 16, 1950, with a history of illness for 24 hours and rapidly progressing spinal paralysis involving all four extremities. Three days after admission some respiratory insufficiency developed which was controlled with the rocking bed. This became ineffective, and an attempt was made to put him in the tank respirator. On the fourth day, owing to excessive mucus and the inability to coordinate with the respirator because of glottic closure, a tracheotomy was done. At this time there was no evidence of pharyngeal, laryngeal, or bulbar signs. No secretions were found in the trachea at the time of tracheotomy. He gradually improved without further repiratory distress, and the tracheotomy tube was removed on the 42nd hospital day, only after he could sustain oxygenation with the use of the accessory muscles of respiration. At this time there was marked respiratory paralysis and paralysis of the extremities and little, if any, cough reflex.

Comment.—Tracheotomy was necessary in this case, or surely the patient would have died of aspiration.

CASE 7.—D. P., a woman 23 years of age, was admitted to the hospital on Aug. 24, 1950. The present illness began four days prior to admission with stiffness of the neck, weakness, general malaise, and slight difficulty in breathing. On the second hospital day slight difficulty in swallowing developed. On the third hospital day she was placed in a respirator because of intercostal and diaphragm weakness. On the fifth hospital day her swallowing became worse, slight cyanosis and listlessness developed, and she was comatose at times. As soon as possible a tracheotomy was done over a bronchoscope, and there was some fluid in the trachea. The lower pharyngeal muscles were paralyzed on both sides, and the soft palate was paralyzed on the right. She improved daily and was removed from the respirator on the 14th hospital day. On the 25th hospital day, while receiving an intravenous solution of isotonic sodium chloride and vitamin B complex, she had a chill, went into shock, and died 12 hours later. Bronchoscopy done shortly before death revealed no secretions in the trachea or bronchial tree. Postmortem examination showed the left lower lobe diffusely atelectatic and not air-containing. There was no evidence of thrombi or bronchial occlusion. The remainder of the lung examination gave negative results.

Comment.—Except for the sudden collapse during an intravenous injection, this patient would surely have lived. Tracheotomy at the time it was done was lifesaving for this patient who had definite signs of anoxia.

CASE 8.—R. S., a boy 8 months of age, was admitted to the hospital on Oct. 9, 1950. On admission the history revealed an indefinite illness of vomiting and a slight fever for two days. The only bulbar finding was an internal strabismus of the right eye; minimal lower extremity paralysis was present. On the second hospital day intercostal and diphragm weakness developed, and he was placed in a respirator. On the third day there was excessive mucus in the throat, and periods of cyanosis developed with some difficulty in swallowing. A tracheotomy was immediately done, and slight secretions were found in the trachea. At this time there was some involvement of the lower pharyngeal muscles. In spite of oxygen therapy, there was little improvement in the condition, with recurring periods of cyanosis. The child died on the 10th hospital day. Postmortem examination revealed few changes in the brain but some in the upper cord. In addition to these there was interstitial pneumonia and an edematous myocardium.

Comment.—There was little indication for tracheotomy earlier. No doubt anoxia secondary to pulmonary insufficiency in the form of bronchopneumonia was responsible for death, with secondary myocardial failure.

CASE 9.—E. E., a girl 8 years of age, was admitted to the hospital on Oct. 26, 1950, with a history of headache and fever for three days. Paralysis developed in the lower extremities the second day in the hospital, and rapidly the upper extremity the intercostals, and the diaphragm became involved. She was placed in the respirator because of slight cyanosis, but at the time there were no bulbar signs. On the seventh hospital day she vomited and aspirated some vomitus which contained little or no solid food. Gradually, over a period of hours, respiratory difficulty became more apparent, and an atelectasis of the left lower lobe developed. A bronchoscopy was done to aspirate the secretions, and then a tracheotomy followed immediately. At this time there were no pharyngeal or laryngeal signs of paralysis. The general condition and color improved. Two days later atelectatic signs on the the left side recurred, and on bronchoscopy 10 cc. of mucoid secretion was removed from the left main bronchus and trachea. On the 58th day decannulation was done after she had been permanently removed from the respirator.

Comment.—Repeated bronchoscopies may have taken care of the secretions and atelectasis, but with tracheotomy management was easier and securer.

CASE 10.—M. T., a girl 15 years of age, was admitted to the hospital on Nov. 26, 1950. The first symptoms began three days before admission, with pain in the neck and difficulty in swallowing. At the time she was first seen in the hospital she was in extreme shock and was

vomiting gastric secretions frequently. A tracheotomy was done immediately, including supportive treatment. At that time there was evidence of pharyngeal paralysis, but the vocal cords moved normally, and there was some mucus in the trachea. The patient died four hours later, having never recovered from shock. A postmortem examination showed marked pulmonary edema with marked evidence of bronchopneumonia in the lower lobes, in addition to marked changes in the brain system.

Comment.—This patient was moribund when first seen, and tracheotomy was of little avail.

CASE 11.—L. S., a girl 2½ years of age, was admitted to the hospital on Dec. 31, 1950. The history revealed that fever and general malaise had developed 13 days before and swallowing difficulty 6 days before which was not constant. Paralysis of the legs and arms began to develop two days before admission, with inability to swallow and frequent attacks of choking. At the time she entered the hospital, all four extremities were paralyzed. Mild cyanosis had developed, with listlessness and marked effort to breathe due to intercostal weakness. Tracheotomy was done immediately over a bronchoscope. There was complete paralysis of the pharyngeal muscles, the larynx functioned normally, and considerable mucus was found in the trachea. The patient never regained consciousness, and several bronchoscopies were done through the tracheotomy opening, revealing each time considerable mucus in the main stem bronchi. On the 9th hospital day she died. A postmortem examination revealed, in addition to findings of poliomyelitis in the cord and brain stem, atelectasis of both lower lobes, including marked congestion and many areas of hemorrhage.

Comment.—This represents another case of marked pulmonary complications probably present on admission but too far advanced to be relieved by tracheotomy.

CASE 12.—S. H., A boy 6 years of age, was admitted to the hospital on May 27, 1951. Three days before, he had symptoms which began with headache, fever, stiff neck, and sore throat. Six hours before admission some difficulty in swallowing developed, and because of progressive respiratory difficulty represented by shallow and irregular breathing a tracheotomy was done. At this time there was complete paralysis of the pharyngeal muscles, there were no secretions in the trachea, and there was good motion of the vocal cords. There was no extremity, diaphragm, or intercostal paralysis. Recovery was steady, and normal deglutition developed on the 14th day. He was decannulated on the 21st day.

Comment.—Tracheotomy undoubtedly was lifesaving in this case and was done at the critical time before marked cyanosis or pulmonary complications had developed.

CASE 13.—G. P., a boy 4 years of age, was admitted to the hospital on June 20, 1951. A sore throat, fever, and stiffness of the neck began eight days before admission, but no difficulty was noted until one day before admission, when fluids came through the nose on swallowing. After admission mucus in the throat became much worse. Direct laryngoscopy showed the soft palate and the lower pharyngeal muscles to be paralyzed. Tracheotomy was done 10 hours after admission over a bronchoscope, and there were no secretions in the trachea. There was slight involvement of the upper extremities, without any of the diaphragm or intercostals. The tracheotomy tube was removed on the 21st day, when he was able to swallow solid foods.

Comment.—Tracheotomy was done at a critical time before there were definite signs of aspiration of secretions.

CASE 14.—J. M., a girl 3 years of age, was admitted to the hospital on June 25, 1951. Fever, general malaise, and stiff neck began three days before. During the first 12 hours in the hospital a good color was maintained, but gradually drowsiness and a slight dusky color developed, with inability to completely remove the pharyngeal secretions by aspiration. A direct laryngoscopy revealed paralysis of the lower pharyngeal muscles, but the vocal cords

showed normal movement. A tracheotomy was done over a bronchoscope, and there were no secretions in the trachea. For two days, except for improvement in color, there was no change in the drowsiness. Improvement was rapid after this, though there was involvement of the right diaphragm and right upper extremity. On the 12th hospital day the patient vomited and aspirated some food which was removed by suction. A bronchoscopy was done later but revealed no secretions in the tracheobronchial tree. Decannulation took place on the 21st hospital day, after she was able to swallow well for several days.

Comment.—Tracheotomy was believed to have been done at the critical time before periods of cyanosis developed.

CASE 15.—H. K., a man 21 years of age, was admitted to the hospital on July 17, 1951. Onset of symptoms began six days before with headache, stiffness of neck, fever, and weakness of the lower extremities and abdominal muscles. Two days after admission he was placed in a respirator because of rapid ascending paralysis involving the upper extremities, diaphragm, and intercostal muscles. Two days later he became irrational, and slight cyanosis developed. A bronchoscopy was done, and copious secretions, were removed from the trachea. An anesthetic polyethylene plastic intubation tube was left in the larynx and upper part of the trachea. At the time this procedure was done, there was weakness of the lower pharyngeal muscles. The minute volume increased from 1.9 to 8 liters per minute. Because of the difficulty in maintaining an airway tracheotomy was done two days later. His condition improved rapidly, and after his swallowing reflex returned to normal and he could remain out of the respirator for long periods, the tracheotomy tube was removed on the 28th hospital day.

Comment.—Frequent bronchoscopies were anticipated, which undoubtedly would have caused a great deal of mucus; so tracheotomy was done finally in preference. The intratracheal polyethylene plastic intubation tube is not considered satisfactory because it apparently caused a great deal of mucus and laryngeal irritation, although the patient tolerated it surprisingly well for over 48 hours.

SUMMARY

A number of patients with bulbar poliomyelitis without spinal involvement were managed by postural drainage and suction. Only 50% of them had swallowing difficulties. Two of the patients in whom tracheotomy was done on admission had severe anoxia presumably due to mucus in the tracheobronchial tree and were moribund. One patient who was not tracheotomized was seen early, but postural drainage did not prevent aspiration. Misinterpretation by the resident staff allowed the patient to progress to asphyxia before appropriate measures could be instituted. Bronchoscopy done at terminal asphyxia revealed the trachea full of secretions.

By far the largest group of those who were seriously ill comprises the bulbospinal patients with breathing difficulties who required a respirator. Of the eight patients who underwent tracheotomy five died. One of this number died accidentally 25 days after admission, presumably from sensitivity to an intravenous injection of vitamin B complex. In the remaining four clinical cyanosis and aspiration of secretions had developed before tracheotomy was done.

Of the seven patients who died without benefit of tracheotomy one died accidentally when a leak developed around the collar and was not discovered before marked cyanosis had developed. In two patients convulsions suddenly developed, and they died in the seizure; one other patient died of a marked hypertension with central involvement. The three remaining patients had marked pulmonary complications at death.

All the spinal patients requiring a respirator recovered, although two were tracheotomized, one because he would not synchronize his swallowing mechanism with the respirator and because large quantities of mucus and saliva developed in the absence of bulbar signs, and the other because of aspiration of a large quantity of vomitus.

In the eight patients who survived the longest use of the tracheotomy tube was 58 days, and the shortest was 16 days, with an average of 44½ days. Although the tube in most instances was closed after the acute phase, it was left in place to give confidence to the patient while reestablishing deglutition. Many of the patients seemed to swallow puréed foods much better than liquids, and when aspiration took place, this food could be easily removed by suction.

There were no complications which could be attributed to the presence of the tracheotomy tube or to the surgery.

CONCLUSIONS

1. A review of the literature reveals unmistakable evidence that death from poliomyelitis in a great percentage of the cases is due to pulmonary anoxia as a result of ventilatory failure. This may be due to respiratory center paralysis, paralysis of the primary muscles of respiration, obstruction of the larynx, either due to a paralysis or due to aspiration of secretions, and pulmonary edema. The pulmonary complications of atelectasis, bronchopneumonia, and marked congestion of the lungs have been the common findings.

2. Recent studies in several instances reveal that there has been a great reduction in the respiratory case fatality rate by doing tracheotomy early to the point of prophylaxis in many patients.

3. A study of my own cases shows a rather high respiratory case fatality rate of 35% in 1950, due in part to a number of late moribund patients received at the center and also to the performance of tracheotomy at the critical time which was usually too late. The patients in 1951 up to the time of this writing were treated somewhat differently. The same indications were kept in mind, but tracheotomy was resorted to on the anticipated complications in severe cases. This procedure decreased the bulbar and respiratory case fatality rate to 10%.

4. Conclusions based on this small series of cases would not be ample in themselves, but together with those cases in Table 1 they constitute favorable evidence.

5. Pulmonary complications and anoxia can be minimized by assurance of a free airway by tracheotomy performed before the patient is compromised.

6. Once pulmonary complications have developed, the value of tracheotomy decreases, especially if marked cyanosis has occurred and especially if a respirator is required. Bronchoscopy is effective in chronic cases with atelectasis, but it does not give the assurance of a continued free airway in the acute cases.

7. Tracheotomy in simple bulbar poliomyelitis is definitely indicated for those patients with adductor paralysis of the vocal cords, especialy those with swallowing difficulty or those showing marked listlessness or coma. Patients with severe swallowing difficulty were materially helped by the tracheotomy tube during the reeducation period of swallowing. Aspiration frequently took place, and suction could easily be carried out.

8. Tracheotomy for bulbospinal patients with respiratory paralysis requiring a respirator is most important, and it is believed that recovery is in direct proportion to the number of tracheotomies done. Tracheotomy must be done very early.

9. For spinal patients with respiratory paralysis requiring a respirator tracheotomy in a selected few cases is most valuable when done early because of failure to coordinate swallowing mechanisms with the phases of the respirator or because of coma due to any cause.

10. A review of the literature shows a few complications as the result of tracheotomy. Very few complications are believed to be due to direct breathing into the trachea. In this series there were no complications attributable to tracheotomy.

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Drs. Russell J. Blattner and William A. Spencer assisted and cooperated in the study of these cases.

NEW MUCOUS MEMBRANE FLAP FOR DACRYOCYSTORHINOSTOMY

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AND
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THIS PAPER is being presented with a dual purpose: (1) to stimulate interest among rhinologists in a surgical technique which they alone are best suited to perform and (2) to describe a method of using the nasal wall of the lacrimal sac as a flap which, it is hoped, will lessen the percentage of postoperative closures of the intranasal ostium.

Eminent ophthalmologists, such as Meyer Wiener [1] and Peter Kronfeld,[2] tell us that the intranasal approach to the lacrimal sac is the method of choice. Their reasoning is that the surgeon who performs the original operation should be able also to care best for the postoperative complications. It is in these complications that the ophthalmologist is at a great disadvantage, because most of his failures are due to closure of the intranasal window. The rhinologist, on the other hand, is equipped and trained to prevent adhesions, remove granulation tissue or obstructing polyps, care for postoperative hemorrhage, treat the margins of the ostium, or reopen it, if necessary.

The physiologic mechanism that closes any surgical opening is the purse-string-like force of a ring of contracting cicatricial tissue. It occurred to us that if a procedure could be devised whereby this "ring" was broken, postoperative closures of the ostium would be much fewer. To accomplish this, it was decided, instead of excising the nasal wall of the sac, to cut it free at its superior, anterior, and inferior borders and then swing it back on its posterior attachment as a flap, which would then be sutured to the mucosa of the stump of the middle turbinate. Thus the mucosal side of the flap would become continuous with the mucosa of the lateral nasal wall and make it impossible for a cicatricial ring to form.

The first case in which we used this procedure was in 1939, but the suturing made the operation difficult. After several further attempts it was discovered that the flap could be held accurately in place with a petrolatum (vaseline®) gauze pack, so the suturing was abandoned.

From the Department of Otolaryngology, University of Oregon Medical School.

Read at the Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, Salt Lake City, April 27, 1952.

1. Wiener, M., and Alvis, B. Y.: Surgery of the Eye, Philadelphia, W. B. Saunders Company, 1939, p. 419.

2. Kronfeld, P. C.: Personal communication to the authors, Oct. 13, 1950.

PROCEDURE

A résumé of the procedure is as follows:

Anesthesia.—The operation is usually done with the use of local anesthesia induced by blocking the supratrochlear, infratrochlear, and lacrimal branches of the infraorbital and anterior internal nasal nerves. A submucosal injection of procaine hydrochloride and epinephrine in the nose is superior to topical anesthesia, as it greatly reduces hemorrhage.

Surgical Procedure.—This is divided into three stages. The only variation consists in doing a partial or complete submucous resection immediately before if the septum is deviated enough to narrow the operative field.

1. The Nasal-Mucosa Flap: Three incisions are made with a pointed Bard-Parker knife or a sharp submucous elevator.

(*a*) One is made from the anterior attachment of the middle turbinate anterosuperiorly, vertical to the bridge of the nose 5 to 10 mm.

(*b*) A similar incision is made from the anterior attachment of the inferior turbinate parallel to the first.

(*c*) The ends nearest the bridge of the nose are connected by the third incision. This outlines a rectangular flap, which is next separated with any thin periosteal elevator, and the part nearest the middle turbinate is pressed downward hard enough to tear it out from under the turbinate a short distance. This procedure makes a hinged flap hanging down over the inferior turbinate. When the operative field is easily seen, the second incision is made closer to the first. The chief function of the broader flap is to give greater visibility to the operative field.

2. Removal of the Lacrimal Bone: The exact position of the anterosuperior margin of the lacrimal fossa is marked with a caliper which we have devised for this purpose. When the outer leg is placed over the medial palpebral ligament, the inner leg marks the corresponding place in the nose with methylene blue.

The bone is then removed with a straight-edged chisel or a long dental burr. The bone is usually thinnest over the lower part of the fossa. In some cases the entire bone can be fractured off in one piece, but even a small opening will admit punches by which the remaining bone may be removed until the sac is entirely exposed. The operator may assist in his orientation greatly during this stage by repeatedly pressing the soft tissue into the bony window with his index finger over the sac externally while watching for movement on the intranasal side. With an angled ethmoid probe the anterior and posterior lacrimal crests may be easily outlined. Great care should be used not to go beyond the posterior lacrimal crest, as orbital fat may present and cause considerable difficulty. Often agger nasi, or anterior ethmoid, cells are opened, but these should not be resected farther than necessary, as most will close without difficulty. If the anterior attachment of the middle turbinate overrides the fossa, it should be removed with a punch.

3. Formation of the Flap from the Medial Wall of the Lacrimal Sac: A No. 1 Bowman probe is now passed through the lower canaliculus, and the nasal wall of the sac is tented into the nose. A longitudinal incision the entire length of the sac is made with a No. 11 Bard-Parker knife on a narrow handle (No. 7) as near

the anterior margin as possible. An incision vertical to the first is next made through the base of the nasal wall of the sac, and another is made through the top. The flap thus formed is turned back, as previously described.

A strand of medium (000) dermal suture is carried through the lower or upper canaliculus into the nose. For this procedure a 20-gauge spinal puncture needle that has been cut back to 30 mm. in length and its point rounded is used. A loop of dermal suture is next drawn into the nose by tying a second strand at its middle to the outer end of the first strand with a single knot and pulling it through. Next a piece of umbilical gauze tape about 3 in. (7.5 cm.) long is doubled on itself, placed in the loop of dermal suture, and then pulled up snugly into the sac by traction on the sutures externally. The ends of the gauze are packed against the lacrimal-sac flap, pressing it against the wall of the middle meatus posterior to the lacrimal fossa. Externally the dermal suture is cut off at the punctum or left attached to the brow with adhesive for 24 hours.

The nasal mucosa that was turned down is now replaced, and that part of it that overlaps the packing in the lacrimal sac is excised. A petrolatum gauze pack is placed between the septum and the lateral nasal wall to hold both the nasal mucosal flap and the packing in the lacrimal sac in place.

Finally, the canaliculi and sac are irrigated with isotonic sodium chloride solution to wash out any clots and to allow the operator to lessen the pressure of the gauze if it is too tightly packed for easy flow of fluid. No definite rule is followed regarding postoperative irrigations. The nasal pack is removed on about the second day and the pack in the lacrimal sac on the 14th day. The patient is then carefully watched for two or three weeks to insure against granulation tissue formation, adhesions, etc.

COMMENT

In our series of intranasal dacryocystorhinostomies, begun in 1945 and previously reported,[3] there now are 106 cases, in 38 of which we used the method described above. In three of these cases the intranasal window has closed postoperatively but has since been successfully reopened.

In several cases in which there has been partial postoperative obstruction to lacrimal flow we recently have used a 22-gauge polyethylene tube which has an outside diameter of 0.965 mm. A piece of this tubing 18 to 20 mm. in length is cut with one end square and the other tapering. A piece of ordinary silk thread is passed through the wall of the tube at the square end with a small cutting needle and tied. The tubing is then passed into the canaliculus with the sharp end nasally until the tubing lies entirely within the canaliculus and protrudes into the nose. The thread only emerges through the punctum and is anchored to the skin with adhesive. This procedure prevents the tube from working into the nose and from injuring the punctum, as the thread is much more pliable. It is believed that bringing the relatively stiff tubing out through the punctum, as described by Veirs,[4]

3. Jones, L. T., and Boyden, G. L.: The Rhinologist's Role in Tear Sac Surgery, Tr. Am. Acad. Ophth., July-Aug., 1951.

4. Veirs, E. R.: Non-Surgical Repair of Strictures of the Lacrimal Drainage System, A. M. A. Arch. Ophth. **47**:71 (Jan.) 1952.

is not only less comfortable for the patient but also damaging to the punctum. We have used this method with some success in strictures of the internal common punctum and in narrowing of the intranasal window. In one case a stricture of the inferior canaliculus developed but was unrelieved, even though the tubing was left in place for over three weeks.

SUMMARY

The surgical technique of the intranasal dacryocystorhinostomy is reviewed, with a description of the formation of a mucosal flap from the nasal wall of the lacrimal sac. Thirty-eight cases have been reported in which this method was used, with good results. In three of these, the intranasal window closed but was successfully reopened.

A method of using polyethylene tubing postoperatively is described.

CYTOLOGIC EXAMINATION

Diagnostic Aid in Malignant Tumor of the Antrum

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CHARLOTTE, N. C.

AS EARLY as 1928, Papanicolaou [1] demonstrated that malignant tumors of the uterus could be recognized by cytologic examination of vaginal fluid. The medical world paid little attention to this observation until his extensive, now classic, studies were published, in 1941.[2] The reluctance with which his work was received has only gradually been overcome by an increasing number of confirmatory reports, particularly by Meigs [3] and Ayre,[4] as well as by many others. The procedure of recognizing malignant tumors from shed cells has now been firmly established as a valuable aid in early diagnosis. In addition to vaginal smears, this method of cytologic examination has been successfully applied to other organs, particularly the urinary tract, stomach, and bronchi.[5]

In principle, the technique can be used for the diagnosis of malignant tumors in all accessible body cavities and orifices. To my knowledge, however, it has not been applied to the paranasal sinuses, although it is evident that it may be of value in recognition of malignant tumors in these areas.

REPORT OF CASES

Four cases are presented in which the diagnosis of malignant tumor was first established by cytologic examinations of antral washings. The diagnosis in each case was later confirmed by biopsy.

CASE 1.—J. W. C., a 45-year-old white man, complained of pain in the left side of the head for three months and double vision for one month.

From the Charlotte Eye, Ear and Throat Hospital.

1. Papanicolaou, G. N.: New Cancer Diagnosis, Proceedings of the Third Race Betterment Conference, Battle Creek, Mich. 1928, p. 528.

2. Papanicolaou, G. N., and Traut, H. F.: Diagnostic Value of Vaginal Smears in Carcinoma of Uterus, Am. J. Obst. & Gynec. **42**:193-206 (Aug.) 1941.

3. Meigs, J. V.; Graham, R. M.; Fremont-Smith, M.; Janzen, L. T., and Nelson, C. B.: Value of Vaginal Smear in Diagnosis of Uterine Cancer: Report of 1,015 Cases, Surg., Gynec. & Obst. **81**:337-345 (Oct.) 1945.

4. Ayre, J. E.: Vaginal Cell Examination as Routine in Diagnosis: Study of Vaginal and Cervical Cytology as Related to Abnormal Growths, South. M. J. **39**:847-852 (Nov.) 1946.

5. Papanicolaou, G. N., and Marshall, V. F.: Urine Sediment Smears as Diagnostic Procedure in Cancers of Urinary Tract, Science **101**:519-520 (May 18) 1945. Papanicolaou, G. N.: Diagnostic Value of Exfoliated Cells from Cancerous Tissues, J. A. M. A. **131**:372-378 (June 1) 1946. Herbut, P. A., and Clerf, L. H.: Cancer Cells in Bronchial Secretions, M. Clin. North America **30**:1384-1392 (Nov.) 1946.

Examination revealed a little mucopurulent material in the left middle meatus. The left middle turbinate was swollen and injected. The left eye was proptosed and displaced upward, and there was tenderness over the left infraorbital rim. The left antrum was hazy when transilluminated.

Irrigation of the left antrum through the inferior meatus was carried out on the first visit. Immediately very bloody fluid returned, and it continued to be heavily blood-stained,

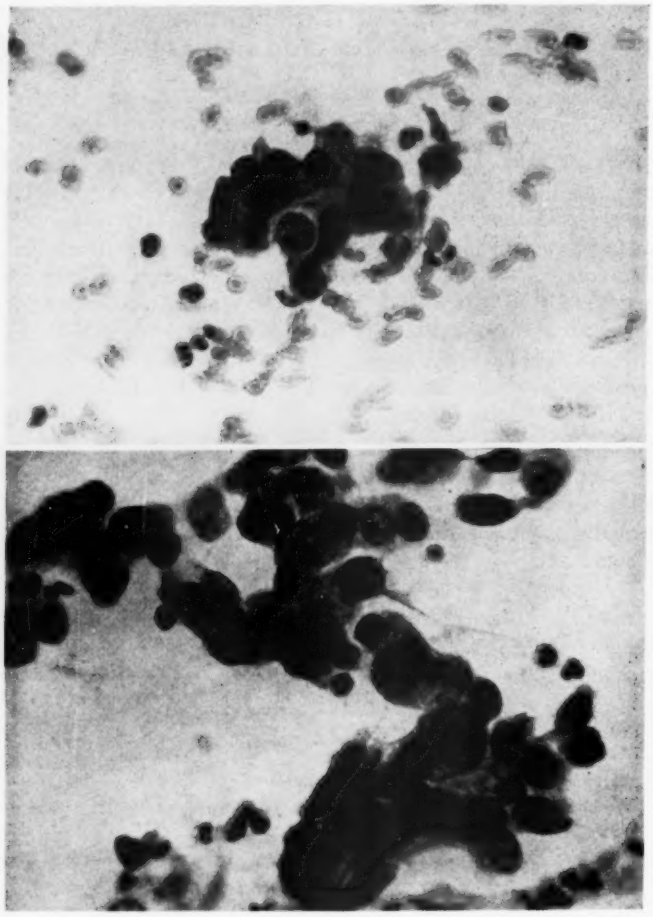

Fig. 1 (Case 1).—Photomicrographs of smear from antral washings (× 600). See text for description of cells.

even after irrigation with 80 cc. of saline solution. The washings were saved to be examined for tumor cells. The cytology laboratory at the Charlotte Memorial Hospital reported that the cells present showed "highly suspicious, but not conclusive, evidence of malignancy." The antrum was again irrigated five days later, and definite cancer cells were found in the washings (Fig. 1). A description of the smear follows: "The smear shows considerable blood and pus. There are many groups of atypical cells. Their rather scant cytoplasm varies in color from bluish-green to purple. The cell membranes are not always clearly recognizable, and, when visible, they appear ragged. The nuclei vary greatly in shape, size, and staining effect

and possess an irregular chromatin pattern. Some of them are finely granular, with scattered large clumps; others are coarsely granular, and still others present themselves as dense chromatin masses. The interpretation is cancer cells." Tissue removed at operation was reported as "carcinoma with features of glandular and stratified squamous epithelium" (Fig. 2).

CASE 2.—L. G. M., a 49-year-old white man, was seen in consultation because he complained of swelling of the roof of the mouth and left cheek, obstruction of the left nostril, watering of the left eye, and double vision. He insisted that these symptoms had been present for only one or two weeks. There was a history of an infection in the left antrum three years prior to his present illness.

Examination revealed the left nasal chamber to be filled with mucopurulent material, but there was no abnormal structure in the nose. A firm mass lay along the inner aspect of the left infraorbital rim and extended to the nose. There was slight proptosis with upward dis-

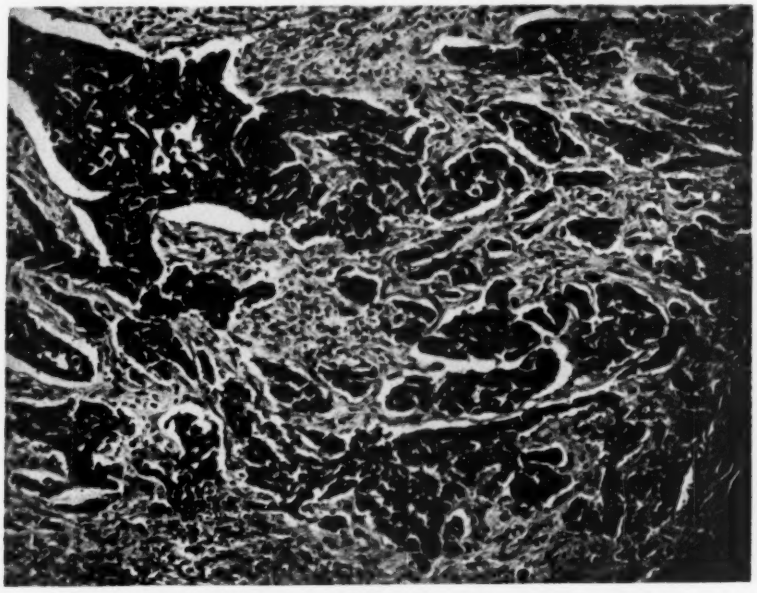

Fig. 2 (Case 1).—Section from surgical specimen, showing closely packed nests of small cuboidal cells infiltrating connective tissue stroma. The nuclei are hyperchromatic.

placement of the left eye and moderate epiphora. A mass was present on the left side of the hard palate. The left antrum was dark when transilluminated.

The left antrum was irrigated through the inferior meatus, and very bloody washings were obtained. The cytologic report follows: "Most of the atypical cells observed in this smear are small. Their cytoplasm is not clearly recognizable. Many of the nuclei are large and irregular in contour, and most of them have a distinct membrane. The chromatin architecture varies from compact clumping to irregular granularity. Several multinucleated elements are seen. One of these cells is very large, ovoid, and finely vacuolar and contains a few dark-blue granules. The nuclei are irregularly shaped with clear borders and a dense, but irregular, chromatin pattern. The cytoplasm of another cell is almost completely taken up by horseshoe-shaped nuclear material. The latter possesses very densely packed chromatin, suggesting in its arrangement the confluence of several hyperchromatic nuclei. The interpretation is malignant tumor cells." (Fig. 3.) Tissue removed at operation was reported by one pathologist as showing reticulum-cell sarcoma; another pathologist reported it as undifferentiated carcinoma (Figs. 4 and 5).

CASE 3.—M. J., a 70-year-old Negro woman, complained of pain of three months' duration in the left side of the face.

On examination a small amount of pus was seen in the left side of the nose. There was no swelling and only slight tenderness in the cheek. Both antra were dark when transilluminated.

The left antrum was irrigated on the first visit, and the unusually bloody washings were saved for examination. The cytologic report follows: "In the bloody and purulent smear there

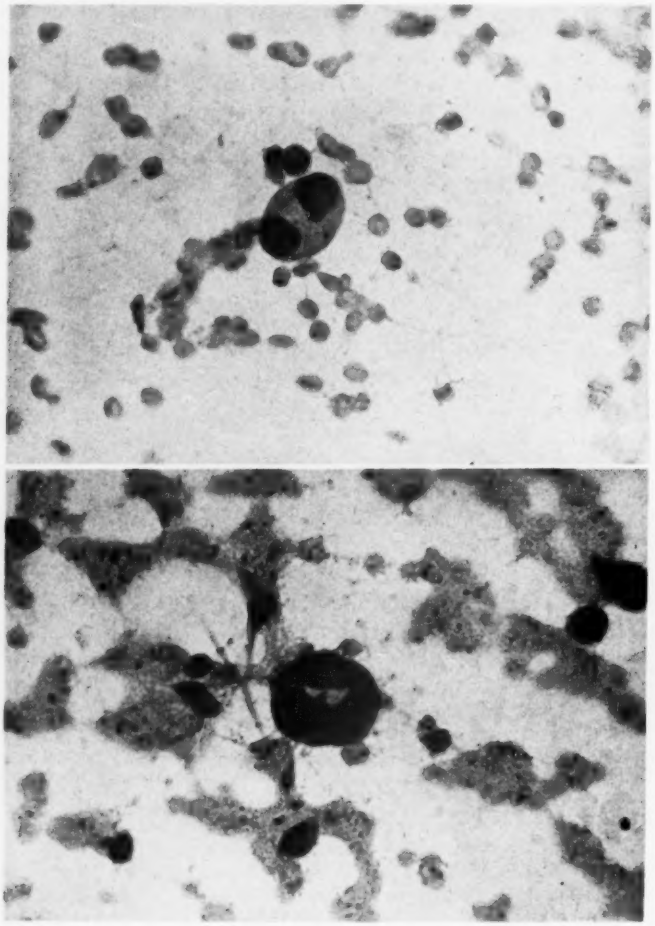

Fig. 3 (Case 2).—Photomicrographs of smear from antral washings (× 600). See text for description of cells.

are many groups of conspicuously large cells, some of which appear almost in sheets. Their cytoplasm is indistinct, often finely vacuolar, and the borders are blurred. The nuclei show great disparity in size, shape, and chromatin density. Many of them are extremely large and appear almost devoid of cytoplasm. The nuclear membranes are distinct, but the chromatin pattern varies from a fine dusty appearance to a coarse granularity and irregular clumping. Most nuclei appear round, but some of them are elongated. The interpretation is malignant tumor cells." (Fig. 6.) A biopsy specimen was taken through an incision in the gingivolabial fold. This was reported as showing highly dedifferentiated carcinoma (Fig. 7).

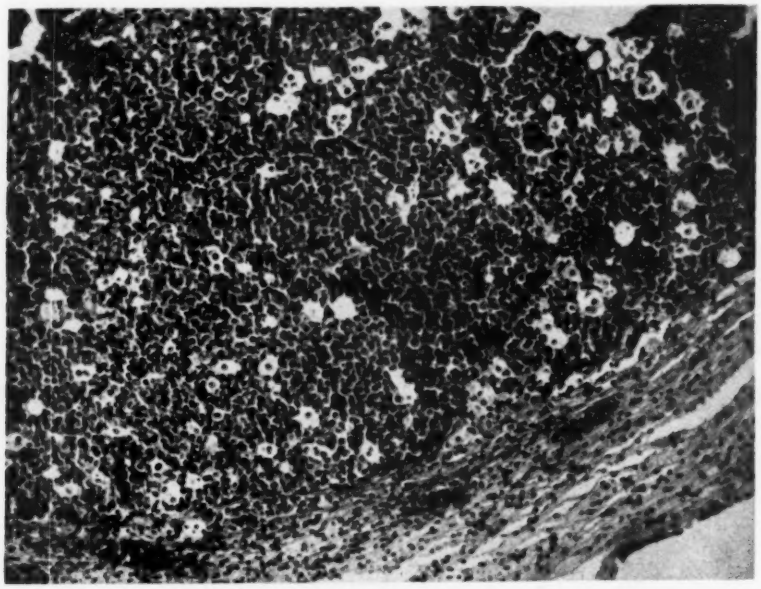

Fig. 4 (Case 2).—Section from surgical specimen, showing large sheets of polygonal cells having no recognizable pattern of arrangement. Low-power magnification.

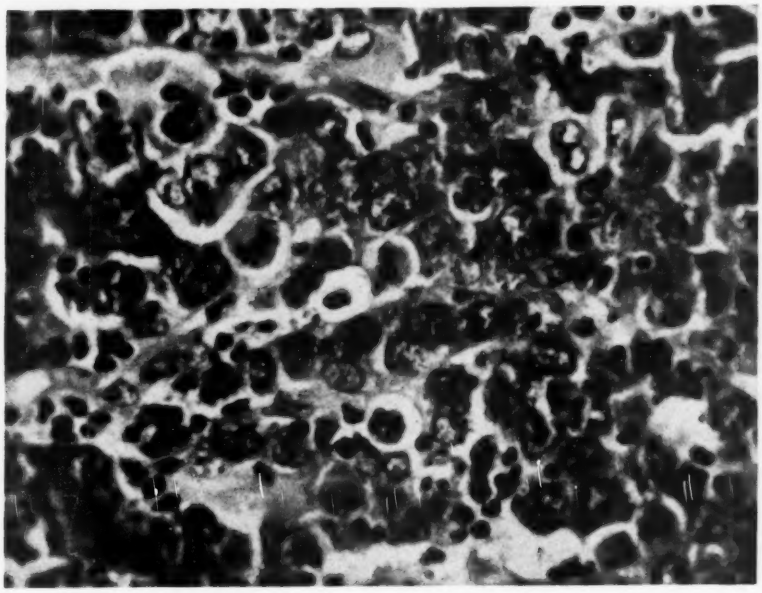

Fig. 5 (Case 2).—Section from surgical specimen, showing large, very irregular cells with poorly stained, and often poorly outlined, cytoplasm. There is only a scant fibrillary stroma. Occasionally the cells are arranged in continuous rows. Cytoplasm as well as nuclei are extremely pleomorphic. Mitoses, including atypical ones, are frequent. High-power magnification.

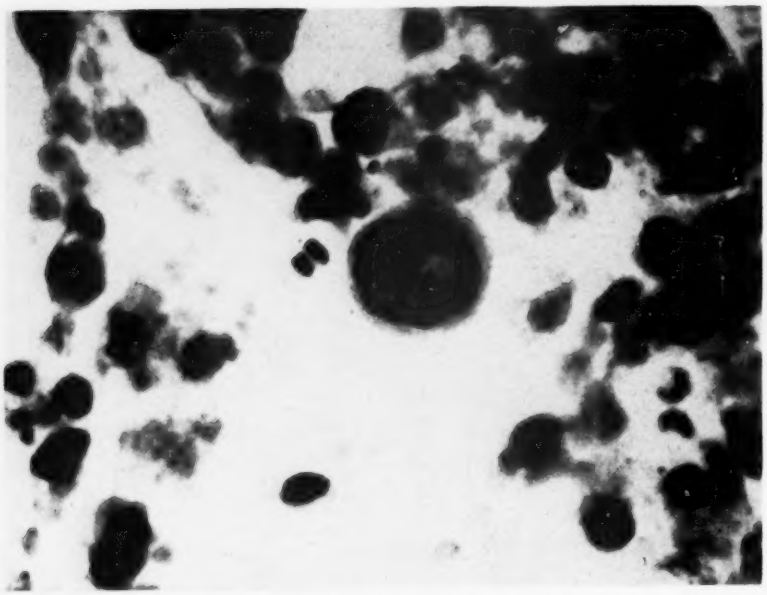

Fig. 6 (Case 3).—Photomicrograph of smear from antral washings (× 600). See text for description of cells.

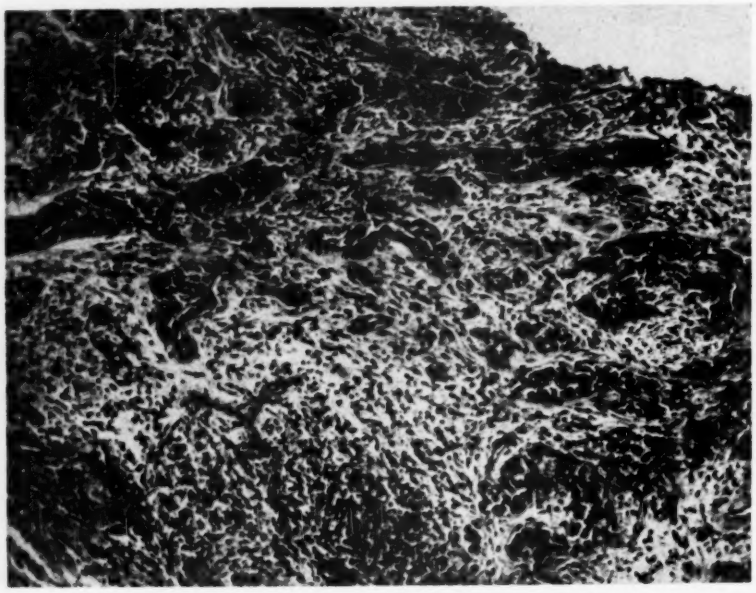

Fig. 7 (Case 3).—Low-power magnification showing nests of epithelial cells infiltrating connective tissue.

CASE 4.—P. W., a 58-year-old Negro woman, complained of pain in the right side of the face, swelling around the right eye, obstruction of the right side of the nose, and dryness of the mouth. All her symptoms had been present for about six weeks and had followed soon after the extraction of some carious teeth. There was no history of previous sinus disease. She had been a known diabetic for 10 years, and for the past 21 months she had been a patient in the North Carolina Sanatorium for the Treatment of Tuberculosis, McCain, with far-advanced pulmonary tuberculosis.

The right nasal chamber was filled with polyps, which originated in the middle meatus. There was slight tenderness over the floor of the right frontal sinus and slight proptosis. There was no evidence of an oral-antral fistula.

With the use of local anesthesia, the polyps were removed, after the antrum was irrigated through the inferior meatus. The polyps were reported as showing "respiratory mucosa with marked chronic inflammation, no evidence of malignancy." The washings, however, contained cells that were regarded as showing highly suspicious evidence of malignancy.

The following day the antrum was again irrigated prior to an intranasal antrotomy. The washings contained definite cancer cells, and the surgical specimen was reported as showing "infiltrating stratified squamous cell carcinoma."

COMMENT

In order to become more familiar with the normal cytology and the cytology in suppurative sinusitis, the antral washings from nine consecutive patients having symptoms of acute or chronic maxillary sinusitis were examined. Superficial squamous cells, columnar epithelium cells, leucocytes, and mucus were found in varying amounts. It should be stressed that considerably more gross blood was observed in the antral washings from the patients with tumor than from those with nonmalignant sinus disease. A large amount of blood in antral washings should arouse suspicion of malignancy.

The first three positive smears were sent to Dr. Papanicolaou for review. In a personal communication,[6] he reported carcinoma in Case 1 (Fig. 1), sarcoma in Case 2 (Fig. 3), and carcinoma in Case 3 (Fig. 6). Examination of the surgical specimens confirmed the diagnosis in each case. Dr. Papanicolaou stated that these were the first instances of malignant growth of the paranasal sinuses that he had observed in which the diagnosis was established by his method.

SUMMARY AND CONCLUSIONS

The usefulness of the cytologic technique as a diagnostic aid in malignant growth of the maxillary sinus is demonstrated in the report of four cases. In each instance, sinus roentgenograms indicated a pathologic condition in the antrum, even to the point of osteolytic changes; nevertheless, the second and fourth cases were interpreted by the roentgenologist as instances of chronic maxillary sinusitis. The diagnosis in the first three cases was probable on the basis of clinical and roentgenographic findings. The potential value of the cytologic technique is illustrated in its application to Case 4, in which the first surgical specimen revealed no evidence of malignancy. Early diagnosis is of greatest importance in the treatment of any carcinoma, and this is particularly true of carcinoma of the antrum. It is believed that the application of the cytologic technique should be as valuable in the early detection of malignancy in the nose and sinuses as it has been in other organs.

Dr. Papanicolaou helped in this study and contributed the photomicrographs appearing in Figures 1 through 5. Dr. Paul Kimmelstiel and members of his staff, particularly Miss Sara Hodges, also cooperated in the study.

6. Papanicolaou, G. N.: Personal communication to the author.

CARDIAC ARREST AND RESUSCITATION FROM VIEWPOINT OF OTOLARYNGOLOGIST

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PHILADELPHIA

CARDIAC resuscitation is not new; in fact, it was attempted on several occasions in the latter part of the 19th century.[1] However, until recently the procedure has been little publicized and little practiced. Beck [2] and Bailey [3] have been pioneers in this field. I am greatly indebted to Beck,[4] who gives an excellent course in the subject under the auspices of the Cleveland Area Heart Association.

Every surgeon should be familiar with cardiac massage and resuscitation. That many lives can be saved by this process is now a matter of fact. No longer may one stand by while a patient dies, assuming that it is an unavoidable and inevitable "anesthetic" death. The lay public is fast becoming educated, through articles in newspapers and magazines, and physicians are being sued for not performing this procedure.

One of the deterrents to practicing cardiac massage is the thought in the surgeon's mind that his chances of bringing the apparently dead patient back to life are poor. Such is not the case. Statistics show that from 33 to better than 50% of the instances of cardiac massage usage are a complete success, but even the latter figure will undoubtedly be raised when there is better knowledge of the procedure. In fact, Beck [5] feels that nearly all, if not all, normal hearts can be resuscitated. Dripps [6] and his associates state: "Young healthy hearts can be restored to normalcy with ease." Krontz [7] illustrated the persistency of the contractibility and the rhythmicality of the heart muscle by reviving hearts (by perfusing the coronary arteries) that had been dead as long as five to six hours, and others are reported as having revived hearts dead for even much longer periods.[2]

1. Barber, R. F., and Madden, J. L.: Historical Aspects of Cardiac Resuscitation, Am. J. Surg. **70**:135-136, 1945.

2. Beck, C. S.: Resuscitation for Cardiac Standstill and Ventricular Fibrillation Occurring During Operation, Am. J. Surg. **54**:273-279, 1941.

3. (*a*) Bailey, H.: Cardiac Massage for Impending Death Under Anaesthesia, Brit. M. J. **2**:84-85, 1941; (*b*) Impending Death Under Anesthesia, J. Internat. Coll. Surgeons **10**:1-10, 1947.

4. Beck, C. S.: Treatment of Cardiac Arrest, Veterans Administration Technical Bulletin 10-65, 1950.

5. Beck, C. S.: Personal communications to author with material from Course on Cardiac Resuscitation, under the auspices of the Cleveland Area Heart Society.

6. Dripps, R. D.; Kirby, C. K.; Johnson, J., and Erb, W. H.: Cardiac Resuscitation, Ann. Surg. **127**:592-604, 1948.

7. Krontz, W. B.: Revival of Human Hearts, Ann. Int. Med. **10**:330-336, 1936.

The incidence of cardiac arrest or cardiac failure is uncertain. Cases are just beginning to be reported. Bailey's [3b] figure of 1 death in every 1,000 anesthesias is probably too high. Beck [5] guesses that there will be five cardiac arrests yearly in a 700-bed hospital.

There are many possible causes of cardiac arrest or failure. These may primarily affect either the cardiocirculatory or the respiratory system. Failure of one system soon results in the failure of the other; hence both must be considered equally.

Anoxemia perhaps stands out preeminently. Wiggin [8] and his associates state, "The most important preventative measure during anesthesia is the maintenance of adequate oxygenation at all times." Lack of oxygen either to the brain centers or to the heart itself (coronary arteries) will cause failure. Various factors may be responsible. The oxygen may not enter the lungs because of an obstructed airway. The obstruction may be from mucus, from a blood clot (tonsillectomy), or from a foreign body, or there may be laryngeal spasm (thiopental [pentothal®] vagal stimulation). The respiratory center may be depressed (morphine sulfate, too deep anesthesia), so that respiration ceases. Anemia, which may be due to hemorrhage, may result in severe anoxemia, even though the oxygen reaches the lungs and the heart is beating. The otolaryngologist often encounters post-tonsillectomy hemorrhage of varying degrees. He knows that anesthesia under these conditions is extremely hazardous. Anoxemia may be heightened by the use of stimulating drugs, for oxygen lack depends upon the supply and demand.[9] Anything that increases the demand increases the lack. The convulsants and central nervous system or metabolic stimulants will do this. These drugs are to be avoided.

Vagal stimulation may cause cardiac arrest directly. This may be the result of upper respiratory manipulation, particularly of the pharynx (including spraying of the throat), larynx, and bronchi, but also of the nasal passages.[10] It is apparent that again the otolaryngologist is particularly vulnerable to this misfortune. Too light anesthesia and anoxemia both predispose the patient to vagal irritation.[8] The use of atropine or scopolamine is strongly recommended for its vagal blocking effect on the sinoauricular pacemaker. Larger doses, e. g. $\frac{1}{75}$ to $\frac{1}{100}$ grain (0.8 to 0.6 mg.) for an adult, even repeated if necessary, are recommended.[11]

Toxins or poisons may cause respiratory or cardiac failure. As has been mentioned, large doses of morphine sulfate paralyze the respiratory center, as may also phenobarbital or cocaine. If there is an idiosyncrasy to a drug, a fatality may result, even when the drug is given in small dosage. The retention of carbon dioxide (as may occur in a closed anesthetic system) is dangerous.

Organic disease of the heart, including that of the coronary arteries or of the lungs, predisposes the patient to cardiac or respiratory failure. In such circumstances resuscitation of any sort may be useless.

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8. Wiggin, S. C.; Saunders, P., and Small, G. A.: Medical Progress: Resuscitation, New England J. Med. **241**:370-378, 1949.

9. Houston, C. S.: Practical Considerations of Clinical Oxygen Lack, New England J. Med. **240**:683-686, 1949. Schmidt, C. F.: Recent Developments in Respiratory Physiology Related to Anesthesia, Anesthesiology **6**:113-123, 1945.

10. Reid, L. C., and Brace, D. E.: Irritation of the Respiratory Tract and Its Reflex Effect upon the Heart, Surg., Gynec. & Obst. **70**:157-163, 1940.

11. Adams, H. D., and Hand, L. V.: Twenty Minute Cardiac Arrest with Complete Recovery: Principles of Prevention and Treatment, J. A. M. A. **118**:133-135, 1942.

When should one start resuscitative measures? When there is no pulse and no blood pressure. The anesthetist can help in determining this. Without blood pressure apparatus at hand, failure of the pulse is enough justification to proceed. A small vessel may be cut to see if there is any bleeding. If the surgeon is in doubt, he should proceed at once, for time is of the utmost importance. The oxygen supply must be restored to the brain in three to five minutes, or irreversible changes result.[12] As Dripps[6] says, "Prompt diagnosis, courageous decision and intervention based on a rehearsed plan of action can prevent fatalities which at the moment seem inevitable."

Mainly because of the urgency involved, it is worth mentioning a few things which the surgeon should not do. He should not take time to listen with a stethoscope, dilate the rectal sphincter, inject the heart with epinephrine,[13] call his associates for an opinion, or take time to sterilize either his hands or the patient. And, as Beck[5] says: "Do not call the fire department." If there is 1 instance of exposing the heart unnecessarily, there must be 100 instances of the reverse. What is more, should the mistake be made, closure is easily accomplished with relatively little fear of mortality or even morbidity.[5]

The approach of choice for this procedure is the direct thoracic one.[14] As contrasted with the subdiaphragmatic or transdiaphragmatic, the thoracic approach exposes the heart to view and also allows direct massage with both hands or with instruments. Seeing the heart aids in distinguishing between standstill and fibrillation. Direct contact massage with both hands is important in order to maintain sufficient blood pressure, a not too easy accomplishment for the inexperienced operator. If the heart is in fibrillation, complete exposure is necessary for defibrillation.

The actual opening of the chest is comparatively simple. However, there should be some preliminary training of the operating room personnel. Some hospitals have printed directions posted in the operating rooms. There must also be an emergency resuscitation tray available; it should contain a scalpel, rib retractor, rib shears, two medicine glasses, 1:1,000 epinephrine hydrochloride solution, 1% procaine hydrochloride solution, isotonic sodium chloride solution, two 10 cc. hypodermic syringes, and two No. 21 4 in. (10 cm.) needles. If the heart is in fibrillation, a defibrillator is necessary.[15] The anesthetist must have the equipment ready for intratracheal oxygen insufflation of the lungs. Squeezing of a rubber bag will accomplish the latter; even mouth-to-mouth insufflation will suffice temporarily, but a mechanical respirator is far superior,[15] especially if the procedure is to be continued for a long period. While the anesthetist inserts the intratracheal tube, the surgeon prepares the chest. An incision is made at about the fourth interspace

12. Wiggin.[8] Weinberger, L. M.; Gibbon, M. H., and Gibbon, J. H., Jr.: Temporary Arrest of Circulation to Central Nervous System: Physiologic Effects, Arch. Neurol. & Psychiat. **43**:615-634, 1940.

13. Some authorities do not agree. Lahey, F. H., and Eversole, V. H.: Differentiation of Hypersensitive Carotid Sinus Reflex and Cardiac Arrest on the Operating Table, Lahey Clin. Bull. **6**:226-230, 1950. Anderson, J. R., and Faust, F. L.: Management of Cardiac Standstill During Otolaryngologic Surgery, Ann. Otol. Rhin. & Laryng. **60**:5-21, 1951.

14. Beck.[5] Dripps.[6]

15. Wolfe, K., and Rand, H. J., III.: Electro-Mechanical Aids in Resuscitation and Anesthesia, Ohio M. J. **46**:39-40, 1950.

(one should not take time to count) from the sternum all the way down to the bed, about 1 ft. (30 cm.) long. There will be no bleeding, for there is no blood pressure. A cartilage is clipped at the sternum above and below, the rib retractor is then inserted, and then the operator's hand is inserted under the heart and presses the latter up against the sternum. At this point the circulation is started, and the emergency is over. If the heart does not start beating almost immediately, the pericardium should be opened, exposing the heart and allowing the operator to use two hands for massage. On seeing the heart one can usually determine whether it is in standstill or fibrillation. A fibrillating heart looks and feels like a wet bag of squirming worms. However, if there is still doubt, now that the emergency is over an electrocardiogram can be taken.

The operator squeezes the ventricles of the heart between his two hands so that the blood is ejected, waits momentarily for them to refill and then repeats the procedure. A rate of 60 to 70 beats a minute can be maintained. The color of the heart muscle itself is a good indicator of the success of the manipulation. The electrodes of the defibrillator, especially if equipped with suction,[15] may be used in place of the hands and at a rate of about 120 a minute will probably produce a better pressure.[16] Some authorities advocate occlusion of the thoracic aorta to increase the flow in the carotids. Johnson and Kirby[16b] found the carotid flow in dogs to be almost doubled by this procedure. Someone should be ready to relieve the operator in a short time, as massage is very tiring.

If the heart is in standstill—i. e., not fibrillating—and has not resumed its normal beat after a few minutes of massage, 4 or 5 cc. of 1:1,000 epinephrine solution should be injected diagonally (to prevent leakage of blood) into the right ventricle. The plunger is withdrawn before injection is done to assure that the needle is in the cavity. Massage is continued. The blood containing the epinephrine must reach the coronary arteries before there will be a change: firmer muscle tone and better color. The normal heart beat may now begin; if it does not, the procedure should be repeated, several times if necessary. Sometimes a little epinephrine dropped on the outside of the heart aids.

If, on the other hand, the heart is in fibrillation, a different procedure must be instituted. First, before the normal heart beat can take place, the heart must be taken out of fibrillation and put into standstill. This is accomplished in the following way: Massage is instituted just as if the heart were in standstill. When the heart becomes pink, electrodes are placed, one on each side of the heart, and 1.5 amp. at 110 volts is passed through the heart for about one-half second. This usually results in defibrillation, from which point the restoration of the normal beat is undertaken as described above. If, however, fibrillation continues, the next step is to inject into the right ventricle 5-10 cc. of 1% procaine hydrochloride solution. The effect of this injection is to block the nerve impulses causing the fibrillation. The procaine shock procedure may be repeated at intervals until the objective is accomplished. Sometimes it is best to add some epinephrine to the procaine. In fact, this should be done if the procaine hydrochloride itself does not suffice, in spite of the fact that pharmacologically the combination does not seem compatible. If a closed anesthetic system is being used, one should be certain that the soda lime is fresh, so that carbon dioxide does not accumulate.

16. (*a*) Beck.[5] (*b*) Johnson, J., and Kirby, C. K.: An Experimental Study of Cardiac Massage, Surgery **26**:472-478, 1949.

After the normal beat has been established, the massage may be discontinued. The pericardium may or may not be sewn. If it is sewn, the stitching should be done loosely, although tightly enough to prevent the heart from herniating through it. Any blood vessels will naturally be tied and the chest wall repaired.

The immediate postoperative period may be critical. An internist and a thoracic surgeon should also be in attendance. The patient must be under constant surveillance. Cardiac or respiratory stimulants may have to be administered as the situation demands. The oxygen tent should be kept near. Mechanical respiration may be necessary. Antibiotic therapy, intravenous administration of fluids, blood transfusions, thoracentesis, and electrocardiogram records may all be needed. During this period there may be febrile responses, muscular rigidity, twitching convulsions, hyperflexion, and varying degrees of coma. Coma lasting more than 12 hours indicates a poor prognosis.

Recovery may be immediate or delayed, or it may be complete or incomplete. It is even possible for the patient to live as a vegetable, but fortunately very uncommon. Almost always there is complete recovery or death. It is worth repeating that the ultimate prognosis depends largely upon how soon the oxygen supply was restored to the brain.

SUMMARY

The causes, prevention, and diagnosis of cardiac arrest and fibrillation are discussed, and the technique of the emergency restoration of the oxygen flow and of the normal heart beat is shown. Prognosis and recovery are discussed. Attention is directed to the importance of this knowledge to the otolaryngologist.

RECONSTRUCTIVE SURGERY OF THE EXTERNAL EAR

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RECONSTRUCTIVE surgery of the external ear—like that of any other part of the body—deals with the repair of congenital or traumatic defects and the reconstruction of congenital or acquired deformities. The aim of any reconstructive operation must be the restoration of form and function. While in some parts of the body restoration of function is the major issue, in exposed parts—particularly in the face—restoration of form is of equal importance. Hence, in patients with defects or deformities of the ear, reconstruction and repair are very often desired.

There are certain ear deformities which can be reconstructed in such a way that the results achieved are very striking and appear to some patients almost miraculous. There are many other deformities in which patient and physician must satisfy themselves with improvement only, a fact that should be recognized and discussed preoperatively. I have particularly in mind the reconstruction of a total or subtotal defect of the external ear, one of the most difficult tasks in reparative surgery. The crux of the problem is the peculiarly twisted shape and flexibility of the ear cartilage, which is covered with a rather thin skin; to provide these conditions by plastic reconstruction has failed so far. Hence, the question arises whether a prosthesis could be used instead. A prosthesis can be made to look like the other ear, but is still difficult to fasten firmly. Many patients would rather go without it than have the embarrassing feeling of the ear falling off.

DEFECTS OF THE EAR

Defects of the ear are either small, subtotal, or total. Of the smaller defects, the holes in the concha need repair mainly for cosmetic purposes. A small hole in the concha can be reconstructed by transfer of a retroauricular double-pedicle flap, which is elevated in stages, as demonstrated in Figure 1. For larger holes, a single pedicle epithelium-lined flap is used, which is also elevated in stages (Fig. 2).

Defects of the rim of the helix and part of the anthelix are reconstructed with a flap from the mastoid region, which is immediately raised and sutured into the defect. The pedicle of this flap is gradually severed after about three weeks and folded upon the anterior part to make the posterior lining (Fig. 3).

Larger defects consisting of half or three-fourths of the upper part of the ear are repaired with flaps from the mastoid region. The details of the technique are outlined in Figure 4. Total defects of the external ear are reconstructed from flaps of the mastoid region, or, if the skin in this area is destroyed, with skin transplanted either from the cervical region or from the median surface of the upper arm (Fig. 4).

Presented before the Otolaryngology Section of the American College of Surgeons, Atlantic City, N. J., Feb. 12, 1952.

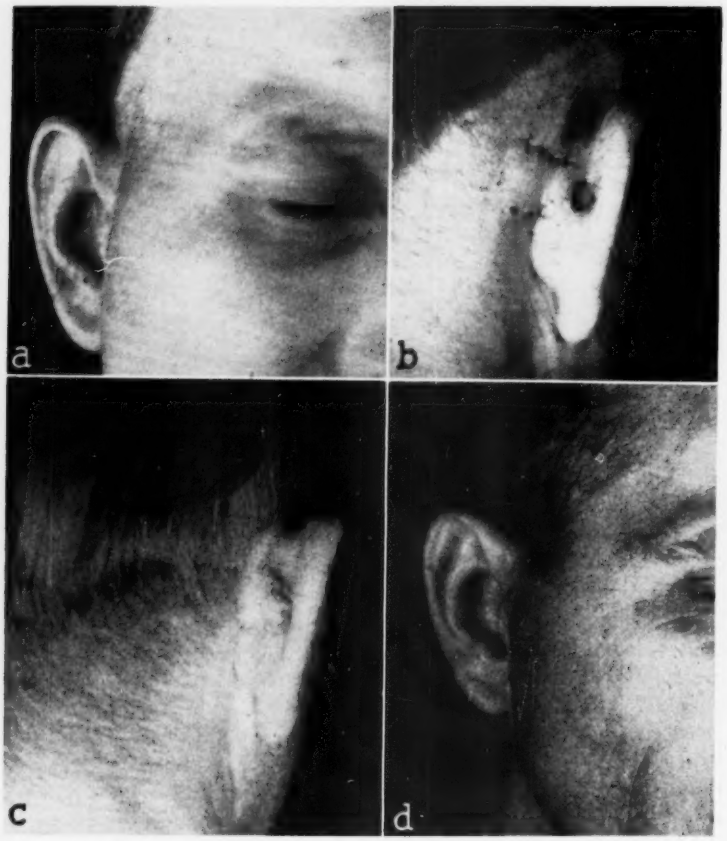

Fig. 1.—(*a*) Bullet wound through concha. (*b*) Double-pedicle flap in postauricular region, one-third larger than required, was elevated and returned. Rim of skin surrounding edge of defect was elevated and returned. This rim of skin was later to be hinged forward to form anterior covering of hole, upon which flap would be laid. After two weeks, pedicle near hole was severed and sutured. (*c* and *d*) After another two weeks, whole flap was raised; rim of skin surrounding edge of hole in concha was hinged forward; auricle was held back, and flap was sutured into defect. After three weeks, backward tilt of ear was overcome by severing central pedicle in mastoid region, and undermining and advancing flap. Resulting raw surface posterior to pedicle was skin-grafted.

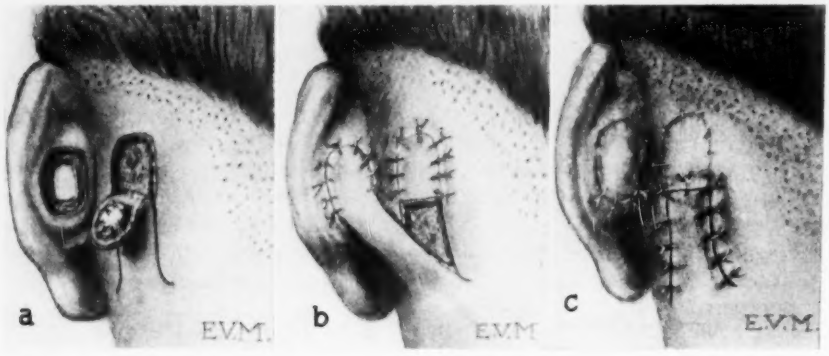

Fig. 2—(*a*) Closure of hole in concha. Single-pedicle flap is raised in stages from hairless mastoid region. Peripheral end is lined with skin graft and flap returned to its original site. (*b*) Flap is raised again, rotated, and sutured into defect. Peripheral end of flap bed is skin-grafted. (*c*) After severance of pedicle and adjustment of pedicle and flap.

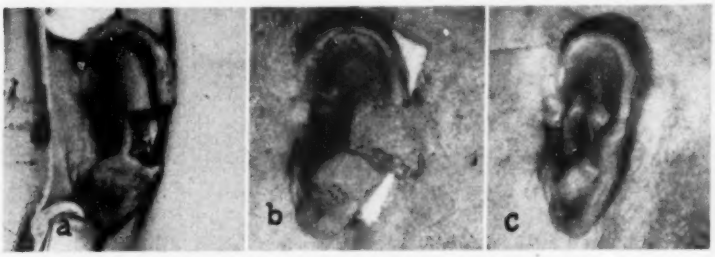

Fig. 3.—(*a*) Partial defect of center part of ear after excision of cancer. (*b*) Immediate repair with flap pedicled in mastoid region. (*c*) Flap had been severed three weeks after transfer, and posterior part of flap was used to complete formation of posterior part of helix.

Fig. 4.—(*a*) Patient, aged 74 years, had squamous-cell cancer at rim of left auricle which had invaded cartilaginous framework. Cancer was removed by subtotal resection of auricle. Reconstruction was begun one year later. (*b*) Flap to be raised from mastoid region was outlined according to pattern and made one-third larger than required. Owing to fact that base of flap came to lie along base of defect—that is, came to lie along a scar—flap had to be raised in stages. Four stages were required, two weeks apart. During first stage, posterior half of flap was depilated. In last stage, rib-cartilage graft, suitably carved to conform with new ear, and diced cartilage, to fill space between latter and base of flap, were inserted beneath flap. (*c*) Ten weeks after last stage, flap and cartilage graft were raised and flap sutured to defect edges. Mold of dental compound was made to hold flap in protrusion, corresponding to that of other ear. Mold was covered with split skin graft, inserted behind ear, held in place with sutures. Mold was removed after ten days. (*d-f*) Patient two months later.

Fig. 5.—(*a* and *b*) Total loss of scalp, posterior part of skin of neck, and left ear. (*c*) Scalp was covered with large split-skin grafts. Ear was built with flap and buried cartilage graft from median surface of left upper arm, and flap was transplanted in stages. (*d*) Six months after operation.

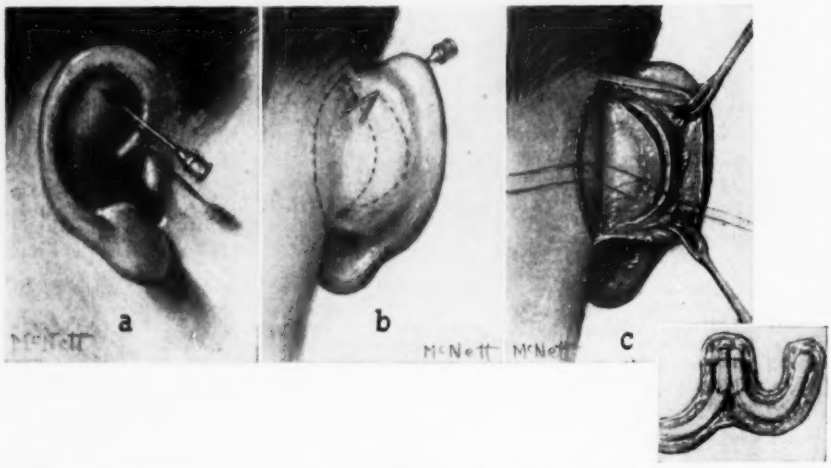

Fig. 6.—(*a*) Protruding ears. Restoration of ridge of anthelix. Ridge of anthelix is marked out by series of punctures made with hypodermic needle dipped in aniline dye. (*b*) Needle is pushed through ear. Before needle is withdrawn, it is touched with aniline dye to stain posterior surface of cartilage. Lateral dotted line is line of punctures or posterior projection of ridge of anthelix. Elliptical piece of skin to be excised is outlined by other dotted line. (*c*) Lateral wound edge is undermined and reflected. Cartilage is divided along line of punctures. Cartilages are folded upon each other (insert) and held together with Lembert-type sutures.

Various methods of replacing the framework are available (Pierce, Gillies, Greeley, Padgett, Kirkham, Peer, Benson, and others); none is ideal. The most difficult part to reconstruct is a sufficiently deep concha; since this ideal in most cases cannot be achieved, it is better to carve the concha from autogenous rib cartilage, or use bank cartilage, and shape to as nearly normal condition as possible.

The details of the operation are as follows: A flap one-third larger than the other ear is outlined in the mastoid region. If the posterior part of the flap comes to lie in the hairy region of the scalp, this region must be depilated. This is best done by shaving the epithelium deep into the corium and applying a thick split graft from a hairless region of the arm or leg. A curved incision along the proposed helix is made. The skin is undermined widely in front of the incision. A piece of cartilage, removed from the fused parts of the seventh, eighth, and ninth ribs, is carved to form a concha, anthelix, and helix, the other ear or a preoperatively made model being used as a guide. The graft is now buried in anatomical position in the previously made pocket. The wound is then closed. A mold of dental compound is now made, with which the skin overlying the cartilage (including the skin-grafted area) is pressed into the convolutions of the graft. A moderate pressure dressing is applied.

After eight weeks, flap and cartilage graft are raised, care being taken not to expose the graft but to leave a sufficient amount of soft tissue upon it. The flap is elevated to the same

Fig. 7.—(*a*) Protrusion of ears due to absence of anthelix. (*b*) After reconstruction.

degree of protrusion as the other ear and is held in this position with a skin graft wrapped around a mold of dental compound.

Dressings, sutures, and mold are removed after 10 days. If the flap shows a tendency to fall backward, a cartilage strut may be inserted at the base of the concha after all wounds have healed. In the absence of the external auditory canal, no attempt should be made to reconstruct it unless coincidental deafness exists.

If the helix is absent, it is replaced by a tube flap about 2.5 cm. in diameter with its base in the mastoid region. It is made long enough to be carried over the ear. Its inferior pedicle is gradually severed after four weeks and transferred and sutured just above the tragus. Three weeks later the base of the tube flap over the mastoid process is gradually severed; the seam of the flap is opened, and the flap draped around the auricle in such a way as to complete formation of the helix.

DEFORMITIES

Protrusion of the external ear is the commonest deformity of the auricle. In the majority of protruding ears the deformity is due to absence of the ridge of the anthelix. The cartilage, instead of folding backward to form the ridge, is flat. The object in correcting the deformity in these cases must be the restoration of the ridge of the anthelix. The second and less common type of protrusion is due to over-

development of the concha, together with an increase of the angle between the concha and head, the anthelix being well developed. The object in reconstruction in these cases must be reduction of the size of the concha and reduction of the angle. The operation for correction of protruding ears is outlined in Figure 6.

If the concha is too large and the protrusion is due to this, an incision is made at the posterior surface of the ear along the sulcus of the cephaloauricular angle. The skin is undermined, and the cartilage of the concha is incised, about 6 to 8 mm. laterally from its base. Under blunt dissection the anterior surface of the lateral part of the cartilage is undermined. This part of the cartilage is now slid over and behind the median part and anchored in this position with sutures to the periosteum of the mastoid process.

CHEWING METHOD AS THERAPY

A Discussion with Some Philosophical Conclusions

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THE SIMPLICITY of the chewing method and the far-reaching conclusions drawn from it present a challenge to look again and again for new insights and to revise our thoughts.[1]

A person who has not paid special attention to the movements involved in chewing knows very little about them. If he is asked to observe the movements closely, he is astonished at the constant movements of the tongue. The tongue has a weak kinesthesia, probably because, unlike the other voluntary muscles, it is only attached to bones at one of its ends. The main factor in orientation concerning a position taken by the tongue is the sense of touch which comes into play if, for example, the tip of the tongue touches the upper incisors or the back of the tongue touches the palate. Yet it seems that even these tactile impressions do not, as a rule, proceed on a highly conscious level. If a slip of the tongue occurs, it is noticed only by the ear. (If the mouth is opened wide and the tip of the tongue is lifted and bent far back without touching the palate, it will be noticed that there is little information concerning the exact position of the tongue. A part of the information will result from the sensation deriving from the stretched frenulum linguae.) Although it is possible that in some dyslalias the "awkwardness" is due to an especially low kinesthesia of the tongue, this does not seem to be true if only one sound in a certain zone of articulation is mispronounced, e. g., in lateral sigmatism. Every logopedist knows how little help he receives, at least at the beginning of the treatment, from the patient's observation of the tongue position. Later the kinesthesia may improve by constant training; yet it is doubtful whether it is not the acoustic self-control which is the real guide.

Since one is limited in self-observation by the weak kinesthesia of the tongue, other proofs of the identity of chewing and speaking movements are always welcome. Recently I had two opportunities to find such proofs. At a dentists' convention [2] Dr. Syrop showed a colored film of the chewing and the speaking movements of the whole mouth of a young man whose entire left cheek and part of the mandible had been removed surgically for sarcoma. The chewing movements of the tongue, both tip and back, were so similar to those of articulation that if food had not been in the mouth one would not have known whether the man spoke or chewed. The second

1. The chewing method was first described in this country by E. Froeschels (Hygiene of the Voice, Arch. Otolaryng. **38**:122-130, 1943).

2. Greater New York Dental Meeting, December, 1951.

opportunity was offered by an x-ray film shown to the New York Society for Speech and Voice Therapy by Dr. Paul Lindenberg. The film showed mouth movements of a subject chewing gum. Not only were the tongue movements again surprisingly similar to those of articulation, but the larynx constantly moved up and down, forward and backward, as is the case in talking. In addition to these observations, another fact caught the interest of the audience, namely, that there was a constant bending and stretching of the upper vertebral column. Here it seems worth mentioning that some dentists as well as logopedists have stressed that chewing as well as speaking and singing should be performed with the vertebral column in an upright position. Dentists have found that a wrong position of the teeth or a malformation of the jaw may have an unfavorable influence on the whole posture of the body, and vice versa. Logopedists could improve the voice by correcting the posture.

In Weiss and Beebe's book, "The Chewing Approach in Speech and Voice Therapy," [3] Weiss has written one chapter on "Chewing and the Origin of Speech." It is my belief that loud chewing, as it still occurs among some African and Australian tribes, is the origin of speech. Of course, when I say that loud chewing is the origin of speech I refer only to the "raw material" used instinctively by the aboriginal human inhabitants of the earth. The development of languages is a quite different topic, and is dealt with by other branches of science, for example, folk psychology, linguistics, and psychology. However, authoritative writers in these fields have by some of their remarks inadvertently given support to this theory. Révész [4] speaks about the material available used by primitive beings for speech. This "material" may well have been the loud chewing. Paget [5] recently wrote about the "gabbling tribes." This expression, I suppose, may also correspond to loud chewing.

It seemed reasonable to look for more clues pointing to the belief that man originally chewed aloud; some can be found in the behavior of small babies and animals. Occasional observations made by laymen have time and again supported my theory of the origin of human speech. I quote here only three of such observations. A lady whose son I treated for stuttering with the chewing method told me, after hearing my explanation, that her cat always purred during chewing. The mother of another stutterer, who also was present during my initial explanation, wrote me the following in a letter: "Long before my youngest son, Frank, was able to talk, I noticed that very often, when he was eating something he particularly enjoyed, he used to hum to himself. Now 12 years old, he still occasionally hums while eating something he likes." The next quotation seems to me to be of great significance: "I want to report to you some experiences which we had as children in our home. We were frequently visited by refugees from Russia and Poland. They constantly produced during eating loud tones which were unpleasant to us. Our

3. The Chewing Approach in Speech and Voice Therapy, edited by Deso A. Weiss and Helen H. Beebe, Basel, Switzerland, S. Karger, 1951.

4. Révész, G.: Ursprung und Vorgeschichte der Sprachen, A. Franke, Bern, Switzerland, 1946.

5. Paget, R. A. S.: The Origin of Language, Science News, No. 20, Penguin Books, Harmondsworth, Middlesex.

requests to omit the sounds were to no avail because they were not aware of the noises. The family agreed, therefore, to imitate their loud chewing and only in this way were we able to stop the unpleasant habit."

Further proof of the identity of chewing and speaking, and to me the most convincing, lies in my experience with three groups of patients in whom chewing aloud, without exception, produces an instantaneous change of voice. These three groups are persons suffering from (1) hyperfunctional voice troubles,[6] (2) mutational (voice-change) difficulties, and (3) voice troubles of the congenital deaf or hard of hearing. The successful application of the chewing method [7] in all of these cases has been described and demonstrated with case histories in several publications, especially in the Weiss and Beebe book. What I should like to offer here is an additional explanation of why such good and astonishingly fast results can be obtained with the chewing method. A therapist who has worked with many other methods before trying the chewing method cannot but observe that the latter works on an entirely different basis. The time necessary to cure the patient will be an impressive point of comparison. In every case the loud chewing works instantaneously, that is, the voice changes immediately into a normal voice. Of course, it takes some time to get the patient used to his new way of speaking or singing.

The final aim of the therapist working with any of these troubles is the same. He wants the patient to have a voice that to the expert's ear shows neither hyper- nor hypo-functional signs, that does not waver between too high or too low a pitch (voice change), that does not provoke any unpleasant feelings in the patient's throat, and that does not lead to fatigue. This voice should conform to the general qualifications of a pleasant voice which, above all, is almost never found in a congenitally deaf person. A number of intermediary steps may occur before the final aim is achieved. These steps also have something in common, regardless of the group to which the case belongs, namely, that the already normal sounding and functioning voice during "nonsensical chewing" may revert to some of the original abnormal signs when the patient is asked to "chew words." This is not surprising, since the patterns developed during the time of voice-abuse can be eliminated only by giving great attention to the chewing idea until the new pattern prevails over the old one. Since the old patterns differ, the intermediary steps will also sound different to the therapist. I have used the chewing method for about 17 years and therefore can refer to a great number of cases in which it has been used. Conclusions drawn from so much experience should be reliable. The fundamental conclusion I want to offer here is based upon the frequent observation of an instantaneous change into a normal voice at the first application of the method. If a voice unhygienically used for many years is immediately replaced by a normal one, the method used to produce such a result must have been somehow latent within the patient.

The method is presented in the following way. First I remind the patient that we can talk and chew food at the same time. The two functions are performed with the same muscles. Since we cannot perform two different activities with the same

6. Hypofunctional voice troubles frequently react favorably to the chewing method. A special article will be devoted to the problems of this disorder.

7. This therapy, as is true of most medical techniques, cannot be learned from books or papers. Observation of the method applied to patients is necessary in order to acquire the correct skill.

set of muscles at the same time, chewing and talking movements of the mouth must be identical. Although we chew with closed lips, primitive people open the mouth and even emit voice while eating. Eating aloud with open lips (and extensive movements of the tongue) sounds like a language, and it is our theory that in this way the human race started talking. The patient is asked to chew as usual with closed lips, but without anything in the mouth, and to observe his tongue, which moves continuously during chewing. Immediately afterward he is asked to chew "like a savage," that is, by opening the mouth and with extensive movements of the lips and tongue. Many people when chewing with an open mouth have the tongue continually on the floor of the mouth. In real chewing the tongue makes a great variety of movements, especially frequent lifting and dropping of the tip and back of the tongue. Only this natural, primitive way of chewing guarantees the setting free of the voice. Then the patient must give voice while chewing. If the chewing is done correctly a great variety of sounds escape through the mouth, not a stereotyped "yum-yum-yum."

The patient should chew with voice production at least 20 times a day, for a few seconds only, but should always avoid doing so mechanically. He should maintain the guiding idea that he has food in his mouth. After a few days, he should read aloud several times a day, for one to two minutes only, but should begin with a voiced "nonsense" chewing. It is explained to him that the different languages are nothing but chewing, and his speech function is therefore fundamentally identical with voiced chewing. In other words, he should maintain the idea of chewing whenever he speaks a language. (Chewing food or gum is not advisable because the patient must learn to rely on just the idea of chewing.)

The chewing method has, of course, nothing to do with swallowing, but may judiciously contain the idea of taking something in. In this way it may help to avert the waste of breath which is a frequent trouble of singers and speakers, especially of those with hyperfunction and hypofunction.

Other methods practice step by step the single functions that are combined into a whole by nature. There are breathing exercises, voice exercises, articulation exercises, and exercises to bring forth certain resonances which somehow must be integrated for the production of flowing speech. Even those methods which do not seem to train single functions first call attention to a single part or parts of the whole speaking and/or singing apparatus. "Drop the jaw"; "flatten the tongue"; "take a deep breath"; "direct the tone up to the hard palate," are a few of the usual instructions to the singing or speaking student. The chewing method has nothing in common with any of these educational techniques. With a single stroke, so to speak, it brings forth a hygienic voice. (This hygienic voice has proved to have all the necessary esthetic qualities.) Neither a partial nor the whole function needs to be trained. It is as though the chewing voice had been waiting to be called for. Waited how long? Thousands, perhaps tens of thousands, of years! Our ancestors evidently gave up loud chewing before history was recorded in written documents.

No one can assume that a function which has not been used by mankind for thousands of years can suddenly be called forth and then appear perfectly intact. There is a theory (Negus,[8] Pressman [9]) that the larynx originally had an exclusively

8. Negus, V. E.: The Mechanism of the Larynx, London, C. V. Mosby Company, 1929.

9. Pressman, J. J.: Sphincteric Action of the Larynx, Arch. Otolaryng. **33**:351-377, 1941.

sphincter function (e. g., lungfish) before voice developed in the evolution of animals. Says Pressman, "When the muscle bands of the lungfish closed, the lower respiratory passages were effectively isolated from the upper. The fish could now without jeopardizing the newly developed lungs open its mouth and swallow." In man the larynx still prevents food from entering the bronchi. The bending epiglottis forms a slope for food to glide backward and downward into the esophagus, and the false vocal cords approach each other, thus closing the entrance into the larynx. Millions of years must have passed since the lungfish was nature's highest representative of animal forms; still the sphincteric function of the larynx comes into play when man swallows food; so this function has been used steadily throughout the years. The voice, on the other hand, has not been used during food chewing for thousands of years and yet it appears in perfection when called forth. It is most improbable that mankind has a memory capable of recalling a function so old and so long neglected, so it seems correct to assume that the chewing voice is still active.

Before explaining what is meant by the above statement, I want to refer to the most amazing "reappearance" of the normal chewing voice, namely, in the congenitally deaf or hard of hearing (third group). This may be observed by anyone working with the chewing method. If he makes the patient understand that he must make vigorous tongue and lip movements and not produce a stereotyped "num-num-num," a normally modulated voice will be produced. It could be suggested that a patient belonging to either Group 1 or 2 might still consciously or subconsciously remember the better voice he had used before his trouble started and therefore improves his voice by chewing. This could not be true of Group 1 because the chewing voice is a normal voice and the patient suffering from a functional voice trouble very likely never used his voice hygienically. As to Group 2, it seems questionable whether the mutational voice is not due to anatomic changes taking place in the larynx. Weiss [10] has answered this question thoroughly, proving that "the overwhelming majority of mutational disturbances have to be considered as functional and treated as such." Furthermore, Fairbanks [11] and his associates could show that in 7 and 8 year old boys and girls the voice may suddenly shift in pitch to the extent of one octave. At this age a hormonic pubescent influence is at least not evident. (In my studies on children's voices [12] I found certain signs in 10-year-old children which might suggest a sex hormonic influence before pubescence becomes obvious.) It is impossible to assume that a normal voice resulting from the chewing method in mutational voice cases may be due to recollection of a premutational voice. The chewing voice of such a patient is that of a young man or a young woman and not that of a child.

In the congenitally deaf or hard of hearing any theory of a recollection of his normal voice as a source of a normal chewing voice is rejected by the fact that I am referring here only to the typically most unpleasant voices of the vast majority

10. Weiss, D. A.: The Pubertal Change of the Human Voice, Folia phoniatrica **2**:126-159, 1950.

11. Fairbanks, G.; Hammon, E. L., and Hammon, J. M.: An Acoustical Study of Vocal Pitch in 7 and 8 Year Old Girls, Child Development **20**:71-78 (June) 1949. Fairbanks, G.: Wiley, J. H., and Lassman, F. M.: An Acoustical Study of Vocal Pitch in 7 and 8 Year Old Boys, ibid. **20**:63-69 (June) 1949.

12. Froeschels, E.: Untersuchungen über die Kinderstimme, Zentralbl. Physiologie **34**:11, 1921.

of such persons. Since, as pointed out above, the persistence of a memory of a function abandoned by mankind thousands of years ago cannot be assumed, there seems to be only one possible conclusion. This conclusion is that the chewing voice is still the speaking voice in the congenitally deaf or hard of hearing, in the patients suffering from hyperfunctional voice impediments, and in those afflicted with a mutational voice trouble (the two latter groups with reference to the statements made above). In other words, in all these cases the chewing voice is still alive and active and is not just a relic that can simply be dug up. No further proof for the statement that the same conclusions hold for all men will be required. (Naturally the idea of the sameness of the chewing and speaking voice in primitive tribes who still chew aloud does not need the hypothesis of a recollection of a function abandoned long ago.)

If the chewing voice is still "alive and active" in man, why then are there so many people suffering from functional voice troubles when no one has a bad chewing voice? There seem to be several reasons for this discrepancy. First of all, chewing most of the time proceeds under very little, if any, emotional strain, while speaking is very frequently the expression of strong emotions and also must frequently fight against strong emotions. I found [12] that first- and second-grade school children, not showing any signs of hyperfunctional voice in conversation, started to overcontract their pharyngeal muscles when asked to read. In imperfect readers the emotions deriving from that task suffice to bring forth hyperfunctions. Secondly, it has been stressed by several writers, especially by Nadoleczny,[13] that hoarseness in childhood may be provoked by having children sing songs which exceed the compass of their voices. Thirdly, since imitation plays a decisive role in speech development a child may instinctively imitate the hyperfunctional (also the hypofunctional) voice of one or more members of the family. The mutational voice troubles, at least as a rule, are not due to imitation. The other two factors just described may also play a contributory role in these troubles, but so far no proof has been found for this assumption. At any rate, it is probable that a voice apparatus misused before the onset of voice change is prone to excessive signs of mutation (excessive wavering between too high and too low pitches) and to permanent post-mutational troubles, such as post-mutational fistula voice (Weiss) and prolonged and permanent wavering. It has been claimed that emotional factors, such as the wish to remain a child, contribute to the maintenance of a high voice (postmutational fistula voice).[13a]

The abnormal voice of the congenitally deaf or hard of hearing child is caused partly by factors different from those so far considered. If hyperfunctional and hypofunctional disturbances develop, an emotional basis may be assumed to occur just as it occurs in normal children, especially if the child shouts a great deal in anger or joy. Kampik [14] made observations on this subject that seem of great significance. He found that congenitally deaf children go through the babbling period as normal children do. In this period, which comes at about the middle of the first year and frequently earlier, the children pronounce some sounds and

13. Nadoleczny, M.: Handbuch der Kinderheilkunde, Leipzig, F. C. W. Vogel, 1926, Vol. 5, p. 179.

13a. Moses, P. J.: Social Adjustment and the Voice, Quart. J. Speech **27**:532-536, 1941.

14. Kampik, A.: Lallen taubstummer Kinder, Blaetter Taubstummenbildung **43**:354-356, 1930.

syllables and evidently enjoy this "game." (Babbling is considered a training for articulated speech; I believe it also serves as a training of the chewing apparatus, all the more since articulated speech comes much later while the intake of solid food frequently starts shortly after or even during the babbling period.) Nadoleczny [13] also confirms the existence of the babbling period in the congenitally deaf, but he says that they babble less frequently and less extensively. Since hearing his own babbling obviously stimulates the normal baby to more babbling, it is understandable that the deaf child, lacking the acoustic stimulation, babbles less than a child who hears.

Under normal circumstances the modulation of the babbling voice very much resembles articulated speech. (I could easily imagine that a babbling infant might be speaking in a language foreign to me.) Why, then, is this resemblance missing in the deaf or hard of hearing child's speech? The answer to this question seems to be crucial and may support or disprove the theory of the identity of the chewing and the speaking voices. On the one hand we have offered the conclusion that the two functions are the same because otherwise the chewing voice would not be ready to "take over" the place of the pathologic voice; on the other hand the normal babbling voice of the deaf child, which is presumably the chewing voice, does not seem to be ready, or at least it is not used, when the speaking voice in the great majority of these cases develops. However, it has been found that if the deaf child is taught to speak with the chewing method his voice becomes surprisingly normal.[15]

There is a trend not to influence the speech of preschool and kindergarten deaf children in the optic-kinesthetic way but just to stimulate spontaneous utterances. This trend seems to be based on the experience that the deaf child has within him a better voice than that brought forth by the optic-kinesthetic methods. This speaks in favor of our assumption that this voice is the chewing voice. The great discrepancy between the babbling voice and the speaking voice of the majority of congenitally deaf persons does not seem to be deeply rooted but seems rather to be the result of inadequate teaching methods. Of course, the absence of acoustic control prevents the congenitally deaf from having as much variety of modulation as a hearing person, because what we consider normal modulation of a speaking voice is, to a certain extent, the result of imitation of the speech of one's family or environment. A deaf child cannot imitate this acquired modulation.

The main question to be answered in this article is whether or not the chewing voice is still working in mankind in the development of speech. The observations and considerations in regard to the three groups of voice impediments discussed seem to rule out a negative answer. On the other hand, the therapeutic experiences described strongly support an answer in the affirmative.

15. The child is asked to chew, and by having him feel for a second the vibrations that the voice of the therapist produces on the chest wall he will understand that he is to chew aloud. A variety of vowels and consonants will appear in this way, and any missing sounds can be taught by the therapist, who chews aloud. The child is then shown how to produce the new sound during chewing so it will be spoken in a natural voice. With methods which do not introduce the sounds via the chewing voice the child's attention is distracted from his natural voice (his chewing voice) by his attempts at articulation and his attempts to imitate the voice of the teacher. Articulation itself will proceed with overstrained (hyperfunctioning) muscles, and this strain will be transferred to the muscles of the larynx. This explanation seems logical when one considers the high, shrill voices of deaf children trained to speak by other methods.

SUMMARY

The chewing method works instantaneously in many voice troubles, changing an abnormal into a normal voice. Three groups are discussed; namely, the hyperfunctional, the post-mutational, and the voice troubles of the deaf and hard of hearing. The effect of the chewing method on the last group is most impressive. Voiced chewing, as still prevalent in primitive tribes, seems to have been common to the whole human race in a low developmental state. The instantaneous effect of loud chewing (chewing method) strongly suggests the idea that the chewing voice is not a "relic dug up" but is still alive and active in the human race. Therefore it is ready for use whenever the patient is asked to chew aloud. The statement that this function is still alive and active supports the theory that eating accompanied by voice emitting was the origin of human speech.

TREATMENT OF TUBERCULOSIS OF LARYNX

Comparative Study of Streptomycin, Paraaminosalicylic Acid, and Tibione

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STREPTOMYCIN has been used long enough to allow a proper evaluation of this antibiotic in the treatment of laryngeal tuberculosis. Paraaminosalicylic acid and tibione (4-acetylaminobenzaldehyde thiosemicarbasone) have been used for a shorter period. Nevertheless, certain conclusions regarding these newer chemotherapeutic agents can also be drawn at this stage.

This is a survey of 136 cases of tuberculosis of the larynx in which streptomycin was used; 21 cases in which paraaminosalicylic acid was used, and 20 cases in which tibione was used. The streptomycin therapy was started in April, 1947, the paraaminosalicylic acid therapy in September, 1949, and the tibione therapy in December, 1949. The first 20 cases in the streptomycin group were reported in a previous communication.[1] The follow-up since that publication has modified some of our original conclusions.[2]

In any study of a new therapeutic agent, controls are of paramount importance. In this study we have had the advantage of observing for a period of 18 years the tuberculous larynges at Sea View Hospital, which has a bed capacity of 1,600 devoted entirely to the treatment of tuberculosis. We have been able, therefore, to compare the more recent therapeutic agents of the past 3 years with the various agents used in the previous 15 years. Since there is no limitation to the period of hospitalization at Sea View Hospital, and since the same staff supervised these cases, adequate comparison of the newer agents has been possible.

PATHOLOGICAL FINDINGS IN TUBERCULOSIS OF THE LARYNX

The pathological data on 1,000 cases collected at Sea View Hospital were summarized by Myerson in 1944.[3] There is a marked similarity between his series of cases (Table 1) and ours in both the distribution and the types of lesions (Table 2).

From the Otolaryngologic Service of Sea View Hospital, Staten Island, and the Otolaryngologic Service of the University of the State of New York, College of Medicine, New York; services of Dr. J. G. Gilbert.

1. Gilbert, J. G.; Aronoff, J. S., and Rosen, D. M.: Streptomycin Therapy in Otolaryngologic Tuberculosis, Quart. Bull. Sea View Hosp. **10**:3-26 (Jan.) 1948.

2. Gilbert, J. G., in discussion on Wallner, L. J.: The Treatment of Tuberculosis of the Larynx with Streptomycin, Tr. Am. Acad. Ophth. **54**:348-349 (March-April) 1950.

3. Myerson, M. C.: Tuberculosis of the Ear, Nose and Throat, Including the Larynx, the Trachea, and the Bronchi, Springfield, Ill., Charles C Thomas, Publisher, 1944, p. 25.

TABLE 1.—*Distribution of Laryngeal Lesions in 1,000 Cases*

	Infiltration	Edema	Ulceration	Tuberculoma	Total
Arytenoids*	202	235	82	5	524
Interarytenoid space	155	28	66	110	359
Vocal cords*	223	17	90	11	341
Epiglottis†	134	82	106	2	324
Ventricular bands*	161	58	79	8	306
Aryepiglottic folds	36	49	7	0	92
Vocal processes	7	0	55	2	64
Ventricle	0	7	0	9	16
Anterior commissure	0	0	1	1	2
Postericoid area	0	0	2	0	2
Subglottic	0	0	1	0	1
Other lesions:					
Papilloma of the epiglottis		1			
Interarytenoid papilloma		1			
Abscess of arytenoid		1			
Fixation of cord:					
Right, 7; left, 13; both sides, 4		24			
Diffuse hyperemia		25			52
					2,083

* One or both structures.
† Ulceration includes necrosis.

TABLE 2.—*Distribution of Laryngeal Lesions in 121 Cases of Tuberculosis of Larynx**

Sites	Infiltration			Edema			Ulceration			Tuberculoma					
Groups	I	II	III	I	II	III	I	II	III	I	II	III	I	II	III
Vocal cords	12	54	47	2	6	6	4	24	12	..	..	1	18	84	66
Arytenoids	5	6	3	11	46	31	..	11	8	..	..	..	16	63	42
Epiglottis	3	3	3	3	3	3	6	4	1	..	..	..	12	10	7
Interarytenoid space	5	9	16	2	5	1	2	12	7	..	..	..	9	26	24
Aryepiglottic folds	2	4	6	5	25	7	..	1	..	..	..	..	7	30	13
Ventricular bands	2	17	12	3	2	4	2	1	9	..	..	..	7	20	25
Ventricle	2	6	3	..	2	..	..	..	..	..	..	..	5	8	3
Anterior commissure	1	2	2	..	..	..	..	..	1	2	4	..	3	6	3
												Total	77	247	183

* Group I consists of 20 cases; Group II, of 59, and Group III, of 42.

TABLE 3.—*Comparison of Involvement of Lungs and Larynx*

	Groups					
	Lung			Larynx		
	I	II	III	I	II	III
Minimal	1	0	0	1	13	14
Moderate	0	8	7	9	11	7
Mildly advanced	0	2	3	3	16	4
Advanced	8	31	25	5	16	8
Far advanced	11	18	7	2	3	9
Totals	20	59	42	20	59	42

We have observed four types of lesions: (1) edematous, (2) infiltrative (including the granulomatous), (3) ulcerative, and (4) the tuberculoma. The commonest sites of the pathology are the posterior thirds of the vocal cords and in the interarytenoid space. The severity of the pulmonary lesions in most of our cases has been associated with a high incidence of pathology involving the arytenoid and aryepiglottic folds. Lesions in these sites are most commonly accompanied by pain and difficulty in swallowing, resulting in inanition and further deterioration of both the pulmonary and the laryngeal conditions.

Generally, the laryngeal pathology varies directly with the severity of the pulmonary lesions (Table 3). However, this is not always the case. At times, we have found a severe lesion in the larynx with only a moderate or slight involvement of the lungs. Moderate or advanced lesions of the larynx are very frequently associated

TABLE 4.—*Questionnaire Submitted to Patients and Supplemented by Staff Notes*

		Date	Sheet No.
Patient's Name	Ward	Streptomycin started	Date
		Streptomycin discontinued	Date
		Reason	

1. How many cups of sputum do you bring up daily?
2. Describe the sputum.
3. How is your voice at present?
4. How has your voice been in the past?
5. Do you have difficulty in swallowing?
   a. Painful swallowing?
   b. Pain referred to ears?
6. Have you had any dizzy spells in the past? If so, describe in detail.
7. Do you have any dizziness at present time? In what direction, if any?
8. Do you stagger when you walk? Past, present. Describe.
9. Have you had any difficulty in hearing in the past? If so, describe.
10. Do you have any difficulty in hearing at present?
    Right ear?
    Left ear?
11. Do you have any ringing in the ears? Have you had any ringing or buzzing previously?
12. Do you have a discharging ear at the present time?
13. Describe briefly any previous ear, nose, or throat trouble.
14. Do you have any difficulty in breathing? Describe briefly.
15. How much do you weigh now?

with involvement of the tracheobronchial tree. However, tracheobronchial lesions may be present without any laryngeal pathology.

METHODS OF STUDY

The patients in this study were segregated for the purpose of maintaining closer supervision. A questionnaire was prepared and submitted to each patient prior to treatment and periodically thereafter (Table 4). This was helpful in collecting and analyzing the data. Caloric and audiometric studies were performed before, during, and after treatment in the cases in which streptomycin was used. The larynges were examined frequently, especially during the early weeks of treatment. In most cases the examination was checked on each occasion by more than one member of the staff.[4] Repeated roentgenograms of the chest and examinations of sputum were routine.

4. Drs. David Rosen, Louis Feit, Herman Sheppard, and Myron Shapiro, of the otolaryngologic service, contributed to this study. Drs. Benjamin H. Ceytlin and M. J. Pond, of the resident staff, assisted in supervision of the patients.

METHODS OF TREATMENT

In the first 20 cases streptomycin was used in the following dosage: 2 gm. daily in six divided parenteral doses for the first week and then 1 gm. daily in similar divided doses until 90 days of therapy were completed. This group will be referred to hereafter as Group I.

Because of the high percentage of toxic effects on the vestibular system in this group, it was decided to determine whether a lower dosage of streptomycin would be as efficacious without causing the same toxic effects. In the next 116 cases, therefore, the dosage used was either 1 gm. or 0.75 gm. of streptomycin daily for a period of 90 days. Group II comprises the 71 cases in which the 1 gm. dose was administered, and Group III includes the 45 cases in which the patient received the 0.75 gm. dose.

Though paraaminosalicylic acid is being used in many cases, only 21 of these cases are being considered in this report, since in the others therapy has not yet been completed. Originally, the patients received 12 gm. daily by mouth in four equal divided doses for a period of 90 days. This period was lengthened to 150 days in some cases in an attempt to obtain better results. More recently, we have been using 24 gm. of paraaminosalicylic acid daily because of the relatively unsatisfactory results with the 12 gm. dose. In passing, it should be noted that the paraaminosalicylic acid used had varying physical characteristics. Some powders were almost pure white (Calco Chemical Co.), others were light and dark gray (New York Quinine Corp.), and one had a pinkish tinge. The textures varied from a fine powder to a rough, gritty, nonuniform powder. The melting points also varied in the different samples. It has also been shown that paraaminosalicylic acid breaks down into metaminophenol on standing. The latter does not have the same bacteriostatic effect on the tubercle bacillus as paraaminosalicylic acid has.[5] Such factors might explain the variable results obtained with paraaminosalicylic acid at different institutions.[6]

The tibione was administered as follows: 50 mg. orally each day for the first week, 100 mg. daily for the second week, and 150 mg. daily thereafter until the 90-day period was finished. The tibione group comprises 20 completed cases.

It is to be noted that another therapeutic agent was often used with the failures in any of the above five groups in an effort to obtain healing, thus allowing for a comparison of different drugs in the same cases. In certain cases in which failure was noted at the end of 45 days, a second drug was used to complete the course of 90 days.

RESULTS OF TREATMENT

General Considerations.—Prior to the advent of streptomycin, paraaminosalicylic acid, and tibione, our experience at Sea View Hospital indicated that a maximum of 15% of the tuberculous larynges healed through the use of rest, diet, collapse

5. Lehmann, J.: Bacteriostatic Effect of *p*-Aminosalicylic Acid and Related Derivatives on Tubercle Bacilli: Experimental and Clinical Studies, Nord. med. **33**:140-145 (Jan. 10) 1947.

6. Augusta Chemical Co., Augusta, Ga., in a personal communication to the authors, stated that any variation from the white color or from the melting point indicates the presence of impurities. These impurities are of three types: (1) isomers, (2) decomposition products such as metaminophenol, and (3) oxidized bodies.

therapy, and local treatment of the larynx. The infiltrative types of lesions responded best to such methods of treatment, while the ulcerative and edematous lesions rarely healed. Furthermore, the time required for improvement and healing in the older series was always a matter of months and years. Generally, healing of the larynx depended on healing of the lungs. The dictum was, "As the chest goes, so goes the larynx."

In the data that follow, the terms "healed," "improved," "primary failures," and "reactivated" are used. By "healed" is meant a total absence of activity, i. e., a disappearance of edema, ulceration, granulation, and even mucosal injection. This healing occurs as a result of fibrosis. The usual end-picture includes a thickening or scarring of the involved sites; consequently dysphonia may not disappear despite complete inactivity of the lesion. Less frequently, the healing process results in a larynx that reveals no evidence of any preexistent legion and is recorded as "normal" in our clinical records. However, for the sake of simplicity, the "normal" larynges are being included in the "healed" group. The "primary failures" represent

TABLE 5.—*End-Results of Laryngeal Lesions After Streptomycin, Paraaminosalicylic Acid, and Tibione Therapy*

	Streptomycin									
	Group I, 20 Cases		Group II, 71 Cases		Group III, 45 Cases		PAS, 21 Cases		Tibione, 20 Cases	
	No.	%	No.	%	No.	%	No.	%	No.	%
Healed	18	90	64	90.2	37	82.2	13	61.9	5	25
Primary failures	0	....	5	7	8	17.8	3	14.3	4	20
Improved	2	10	2	2.8	0	....	5	23.8	11	55
Healed, then reactivated	2	10	11	15.6	7	15.5	5	23.8	1	5
Summary										
Failures		20		25.4		33.3		61.9		80
{ Primary failures Improved Healed, then reactivated										
Healed (at last examination)		80		74.6		66.7		38.1		20

those cases in which practically no change was revealed, either subjectively or objectively, at the end of therapy and during subsequent follow-up. The "improved" cases lie between the "healed" and "primary failure" cases. Because of their poor prognosis, the "improved" larynges could have been properly placed with the "primary failures," although they have been listed separately in some of the tables. The "reactivated" cases are those in which the larynx was healed at the end of the treatment period but subsequently revealed a recurrence of lesions.

Group I.—In these 20 cases a dose of 2 gm. of streptomycin was used daily for one week and then 1 gm. daily for the remainder of the 90-day treatment period. These cases were followed for varying periods up to 36 months. As can be seen in Table 5, healing resulted in 18 of the 20 cases (90%) and in 2 cases (10%) there was improvement. Of the 18 cases in which healing was effected, 2 cases (10% of the entire group) showed reactivation, 1 occurring seven months and the other two months after cessation of therapy. Thus, there was healing in 16 (80%) of this group of 20 cases while under observation.

Group II.—In these 71 cases the dosage was 1 gm. of streptomycin daily for 90 days. These cases were followed for varying periods up to 30 months. Of this

group healing resulted in 64 cases (90.2%); improvement was shown in 2 cases (2.8%), and 5 cases (7%) represented primary failures. Of the 64 cases in which healing occurred 11 (15.6% of the entire group) showed reactivation, leaving 53 cases (74.6%) in which healing has resulted up to the present time. This figure compares closely with the 80% result in Group I.

Group III.—The dosage in these 45 cases was 0.75 gm. of streptomycin daily for 90 days. These cases were followed for varying periods up to 24 months. In this group, healing was shown in 37 cases (82.2%) and in 8 cases (17.8%) the treatment resulted in primary failure. Of the 37 cases in which healing resulted, 7 (15.5% of the entire group) showed reactivation, leaving 30 cases (66.7%) showing a healed condition to date. It therefore appears that the 0.75 gm. daily dose is not quite as effective as the 1 gm. dose.

It should be noted that of the 119 cases in which primary healing was aided by streptomycin only 20 (16.8%) showed reactivation. What is more, of those in which the healed condition remained for nine months after cessation of treatment, few broke down.

TABLE 6.—*End-Results of Treatment of Laryngeal Lesions on Three Dosages of Streptomycin plus Paraaminosalicylic Acid*

	Dosages			
Healing Period in Months	2 Gm. Daily 7 Days; 1 Gm. Daily 83 Days Group I, 20 Cases	1 Gm. Daily 90 Days, Group II, 71 Cases	0.75 Gm. Daily 90 Days, Group III, 45 Cases	12 Gm. Daily 90-120 Days PAS, 21 Cases
3-6	5	29	17	13
7-12	8	19	17	..
13-18	1	11	8	..
19-24	3	10	3	..
25-30	1	2	..	..
31-36	2	..	..	..

Paraaminosalicylic Acid Group.—In these 21 cases the daily dosage was 12 gm. of paraaminosalicylic acid orally for a period of 90 to 120 days. The cases were followed for varying periods up to 10 months since termination of the treatment. Of the 21 cases, healing was shown in 13 (61.9%), improvement in 5 (23.8%), and primary failure in 3 (14.3%). Of the 13 cases in which healing resulted 5 (23.8% of the entire group) showed reactivation, leaving 8 cases (38.1%) that show healing at the present time. It must also be recognized that this low percentage of cases which show healing will probably be reduced even further as our period of observation is prolonged.

Tibione Group.—Though the period of observation with this drug is very short, we are submitting the data as of June, 1950, on 20 cases. These cases have been followed for varying periods up to seven months since the termination of treatment. The treatment consisted of the oral administration of 50 mg. of tibione daily for the first week, 100 mg. daily for the second week, and 150 mg. daily thereafter until the 90-day period was finished. This resulted in primary healing of 5 cases (25%), primary failure in 4 (20%), and an improved condition in 11 (55%); reactivation

in one case (5% of the entire group, or 20% of those cases which showed healing) leaves only 20% in which healed conditions were found at the time of last examination.

Combined Therapy.—To obtain some information as to the possible synergistic action of the three drugs, the various possible combinations of the drugs are being used in the therapy of some cases. No final conclusions can be made as yet.

Re-Treatment of Failures.—Included under the heading of failures are the "primary failure," "reactivated," and "improved" cases. Re-treatment of this group allowed comparisons of the effectiveness of each of the various drugs and of the various dosages of a single drug in the same patient. This aspect of the study permits an evaluation of the various drugs which is in many ways superior to that derived from the results initially obtained with any single drug. This aspect of the treatment of laryngeal tuberculosis has been largely neglected in the reports of other investigators. For the present we are submitting the following data.

In the cases showing reactivation after the use of streptomycin, a second course of streptomycin or a course of paraaminosalicylic acid caused healing in more than 50% of the cases. However, six "primary failures" and "improved" cases in which streptomycin was used originally in the therapy showed no response to a second course of streptomycin or to the subsequent use of paraaminosalicylic acid or tibione.

In two cases in which paraaminosalicylic acid was used (one a "primary failure" and the other "improved"), a second course of paraaminosalicylic acid failed to improve the laryngeal status. However, of five "primary failure" and "improved" cases in which paraaminosalicylic acid was used originally, in four cases healing occurred with a course of streptomycin. It is interesting to note that the 80% healing in this small group compares favorably with that obtained when streptomycin was used as the initial medication.

It appears therefore from the above report of this small experience with re-treatment of "failures" that the following conclusions can be drawn: 1. In "primary failures" and in cases that show improvement following the initial use of paraaminosalicylic acid or tibione, healing can be effected by the use of streptomycin; 2. in failures occurring after the use of streptomycin, healing cannot be effected by the subsequent use of paraaminosalicylic acid or tibione.

RESISTANCE IN STREPTOMYCIN THERAPY

It has been generally assumed that the laboratory report of high resistance of the tubercle bacillus to streptomycin in a particular sputum indicates a diminished therapeutic value of the drug. Our experience makes this belief questionable for the following reasons: 1. We have seen instances in which the significant improvement occurred in the second half of the 90 days of treatment; yet many institutions have limited therapy to 42 days because the laboratory data have indicated a high resistance to the drug at that time. 2. We have successfully administered a second course of 90 days' treatment in the cases in which reactivation occurred. In several of these cases in which a second course of streptomycin was used in spite of a report of high resistance (100 to 1,000 γ per cubic centimeter) there was a successful outcome. In a few of these cases not only did the larynx heal but the sputum became negative.

TABLE 7.—*Neurotoxic Effects*

Case No.	Sex	Age, Yr.	Auditory Effects: Audiometry: Before Therapy	Auditory Effects: Audiometry: After Therapy	Auditory Effects: Tinnitus	Vestibular Effects: Caloric Test: Before Therapy	Vestibular Effects: Caloric Test: After Therapy	Vestibular Effects: Clinical Manifestations	Comment
					Group 1 (20 Cases)				
1	M	40	Normal	Normal	None	Not done	No response of left labyrinth after 1 mo. of therapy; persisted; right labyrinthine response normal	No vertigo; very slight involvement of gait on rough surfaces observed after completion of therapy	.....
3	M	45	Loss in high tones (i. e., 2048 cps and above) of 20 to 30 db. especially in left ear	No change up to 2048 frequency; at 2048, drop to 45 db. and 4096 a drop to 75 db. bilaterally	None	Normal	Normal	No symptoms; normal gait	.....
4	M	61	Patient deaf in both ears for 4 yr.	Possible increase of deafness following therapy	Present between 2d and 6th wk. of therapy	Lightly depressed response in left ear	No change	Slight dizziness and involvement of gait	Difficult to evaluate audiometrics and subjective symptoms because of extreme language barrier and pretherapy deafness
5	M	59	Average loss of 20 db. of left ear and 40 db. of right ear	No essential changes	Occasionally during 6th wk. of therapy	Left labyrinth active	Left labyrinthine reactions absent after 4 wk. of therapy and remained so	Dizziness present between 2d and 8th wk.; too weak for performance tests	Chronic suppurative otitis media (right ear) became inactive during therapy and has remained dry
9	F	33	Not done	No significant loss	During first 2 wk.	Not done	Left active responses; right sluggish responses with inward past-pointing on both vertical and horizontal positions	Dizziness during first two wk.	.....
11	F	28	Normal	Normal	None	Normal	Normal	Some vertigo on change in position during first 10 wk. of therapy; slight ataxia persisted after completion of therapy	.....
12	F	26	Normal	Normal	None	Normal	Both labyrinths inactive	Marked vertigo and staggering present during first 6 wk.; thereafter, slow improvement was noted, though some involvement persisted even after cessation of therapy	.....

13	F	29	Normal	Normal	None	Normal	Normal	Slight vertigo and staggering present during 2d wk. for 3 or 4 days	.....
15	M	27	Not done	Normal	None	Not done	Normal	"Dizziness" experienced before, during, and after therapy	Patient adjudged to be severely psychoneurotic by all attending physicians
16	M	40	Not done	Not done	None	Not done	No response from either labyrinth	Dizziness and staggering gait occurred in 2d wk.; symptoms started to subside 4 mo. after therapy; 5 mo. after therapy, there was no vertigo and only minimal staggering	.....
17	M	56	Bilateral loss in high tones (i. e., from 1024 db. up)	No significant change	None	Not done	Both labyrinths inactive (left ear tested with 5 cc. of iced alcohol)	Dizziness and drunken gait appeared on 20th day and persisted after therapy; both are present to slight degree 5 mo. after therapy	Patient had a tuberculous otitis media of left ear before therapy; this ear became dry during therapy but started to discharge again 4 mo. after cessation of therapy
19	M	37	Normal record	Air conduction dropped to 70 db. at 4096; no hearing with bone conduction at 4095	None	Normal	Normal	No symptoms	Patient received a second course of streptomycin for genitourinary tuberculosis
					Group 2 (39 Cases)				
91	M	50	Normal record	Normal	None	Normal	No response from either labyrinth	Dizziness starting in 3d wk.	.....
					Group 3 (42 Cases)				
13	F	40	Normal record	Normal	None	Normal	No response from both labyrinths 1 yr. after therapy	Slight dizziness 2d wk. of therapy	.....
61	F	52	Normal record	Normal	None	Normal	No response from either labyrinth	Dizziness after 2d wk. of therapy	Dizziness subsiding 1 yr. after therapy

We believe the resistance tests routinely performed in the laboratory are inadequate and may lead to erroneous deductions, since they test only the particular sputum that is being coughed up at any one time. This sputum could come from an open cavity while at the same time other tubercle bacilli are trapped in closed-off spaces. It is our feeling that the resistance of tubercle bacilli may vary greatly in the several cavities in the same patient.

Therefore we are using a new approach in the study of streptomycin resistance, in conjunction with the surgical service. In cases in which the therapy has included both a 90-day course of streptomycin and excisional surgery, tubercle bacilli isolated from the several cavities in the excised specimen are being studied to determine their individual levels of resistance. In cases in which tuberculous empyemas as well as cavitations in the lung are found, and which result in pneumonectomy, a comparison is made between the resistance levels of the tubercle bacilli found in the sputum, in the intrapulmonary cavities, and in the pleural space. This study will be the subject of a separate report from the otolaryngological and surgical services.

It is generally assumed that the combination of paraaminosalicylic acid and streptomycin is superior to either drug alone, especially from the viewpoint of preventing increased resistance of the tubercle bacillus. We feel, however, that this will remain an unproved point until sufficient clinical data are obtained from cases in which the two drugs are being used simultaneously and the results are compared with the data obtained from the use of each individual drug. This study is being carried out at the present time.

Toxicity.—We are concerned only with the neurotoxic effects of streptomycin on hearing and balance. Since the first report of such untoward reactions, by Brown and Hinshaw,[7] these have been generally considered to be the most important complications in the use of streptomycin. During these past 36 months, evidence has accumulated that highlights two conclusions: Vestibular effects are frequent and auditory effects are relatively rare. This is unusual since the vestibular system, because of its greater age phylogenetically, has generally been found to be more resistant to noxious stimuli (drugs, infection, etc.) than the so-called younger auditory apparatus. Exceptions to this rule are few; they include epidemic encephalitis, botulism, and, rarely, syphilis. Among drugs, only the barbiturates appear to depress vestibular function selectively. The pathological basis for the neurotoxic effects of streptomycin has now been determined to be due to a degeneration of the hair cells in the semicircular canals and the organ of Corti.[8]

Table 7 reveals that none of the 136 patients treated with streptomycin revealed a marked loss of hearing and only a few possibly showed a slight loss. The toxic effects on the vestibular system were common (40%) in Group I, in which the initial dose was 2 gm. daily for the first week and 1 gm. daily for the rest of the 90 days. In Group II the dosage was 1 gm. daily for 90 days, and only 1.6% revealed any effects on the vestibular system. In Group III the dosage was 0.75 gm. daily for 90 days, and 4.7% revealed some toxic effects. It therefore appears that daily

7. Brown, H. A., and Hinshaw, H. C.: Toxic Reaction of Streptomycin on the Eighth Nerve Apparatus, Proc. Staff Meet., Mayo Clin. **21**:347-352 (Sept. 4) 1946.

8. Hawkins, J., and Causse, R., cited by Glorig, A.: The Effect of Dihydrostreptomycin Hydrochloride and Sulfate on the Auditory Mechanism, Ann. Otol. **60**:327-335 (June) 1951.

doses above 1 gm. for even a short period (seven days) markedly increase the frequency of toxic symptoms. But prolonged administration (up to nine months or more) did not cause increased toxicity so long as the dose did not exceed 1 gm. a day. This does not coincide with the opinion of the Veterans Administration to the effect that the severity and frequency of all forms of toxicity increased with an increase of the total dose.[9]

It is also to be noted that if the toxic symptoms did not appear within 30 days of the institution of treatment the chance for toxicity to appear at all was practically nil.

Paraaminosalicylic acid has caused only gastric irritation. This can be prevented by the administration of the drug in cold tomato juice or ginger ale.

Tibione has the following toxic effects: development of leucopenia, severe secondary anemias, exfoliative dermatitis, and toxic hepatitis with vomiting. At Sea View Hospital, in 20 cases on our service, leucopenia developed in one instance, and a very severe secondary anemia developed in a second. In both cases the administration of the drug was halted. Toxic hepatitis with vomiting developed in two others. On the medical service, of 85 cases in which the drug was used leucopenia occurred in one instance. Frequent blood counts are needed during tibione therapy. Recently, in one case in which tibione had been used for 47 days, severe exfoliative dermatitis developed over the patient's whole body. Tibione was withheld from the patient at this point, and paraaminosalicylic acid was subsequently used (24 gm. daily), with no return of the skin rash. According to the report of McDermott and Hinshaw,[10] the German investigators reported a 0.4% incidence of agranulocytosis and no cases of exfoliative dermatitis. The toxic effects we observed occurred despite the fact that our daily dosage was one-half that used by the Germans.

COMMENT

In the past, improvement of the tuberculous larynx depended on improvement of the pulmonary disease. The dictum was, "As the chest goes, so goes the larynx." This is no longer true.

With streptomycin therapy, the larynx has healed independently of any change in the lungs. Usually little change in the condition of the lungs has been observed in roentgen studies, while the larynx has healed. In several instances, while the larynx improved and eventually healed, the pulmonary disease was progressing and led eventually to death. It should be noted, however, that in those cases in which dysphagia developed (those with involvement of the arytenoids and aryepiglottic folds) there was an early amelioration of this distressing symptom following the use of streptomycin. This is followed by improved nutrition, gain in weight, and better mental state. In some of these cases, this general improvement has also been reflected in a subsequent improvement of the chest condition and a conversion of sputa from positive to negative. Most significantly, the healed larynx permits the use of prolonged endotracheal anesthesia. This allows indicated excisional pul-

9. Minutes of the Seventh Streptomycin Conference, U. S. Veterans Administration, Denver, April 21-24, 1949.

10. McDermott, W., and Hinshaw, H. C.: A Summary of Observations Made in Germany on the Use of Conteben (TBI-698) in the Treatment of Tuberculosis, New York, Schenley Laboratories, 1949.

monary surgery, which heretofore was impossible with active tuberculosis of the larynx. In 13 such cases (to be reported by the departments of surgery and otolaryngology), surgery on the lung was made feasible. In these instances, the old dictum could well be reversed to read: "As the larynx goes, so goes the chest."

Of the three therapeutic agents, streptomycin has proved to be far more valuable than either paraaminosalicylic acid or tibione. It has resulted in the greatest percentage of cases with primary healing. The improvement has generally been far more rapid with streptomycin, and so the use of local analgesics and superior laryngeal nerve block is no longer necessary (Table 8). Most important, in cases in which streptomycin has been used the healed status has thus far been maintained for the longest periods and the lowest percentage of reactivations has obtained (Table 5). In the 119 cases in which streptomycin was used and in which primary healing was indicated, there were only 20 reactivations (16.8%). Of these 20 cases, 17 reactivations occurred before nine months had elapsed after treatment was discontinued. It appears, therefore, that reactivation is not likely to occur if the larynx has remained healed for nine months following termination of therapy.

TABLE 8.—*Onset of Subjective and Objective Improvement After Beginning Treatment with Streptomycin*

	Subjective Improvement in Groups			Objective Improvement in Groups		
Time Noted, Days	I	II	III	I	II	III
0-7	2	2	7	0	1	7
0-14	9	26	10	6	18	18
0-21	2	22	13	4	21	10
0-28	5	4	3	6	12	0
0-35	0	3	0	1	3	0
0-42	0	0	1	2	1	1
0-49	2	0	0	1	1	0
No change	0	2	5	0	2	6
Totals	20	59	42	20	59	42

In spite of only limited or no improvement shown in the roentgen studies of the chest, many of these streptomycin-treated patients are so improved that they have returned home and are engaged in useful pursuits. Others are employed in limited occupations in the hospital rehabilitation program, while most of the bed patients are now ambulatory. Without attempting to minimize the value of these roentgen studies, we feel that they do not always afford a complete perspective of the patient's clinical condition.

Paraaminosalicylic acid has been of value but not nearly of as much value as streptomycin. Not only have we observed a lower percentage of cases of primary healing, but the number of reactivations is increasing as the period of observation continues. The total of failures ("primary failures," "improved," and "reactivated") was 61.9%, compared with 25.4% in which the dosage of streptomycin was 1 gm. daily. At the present time we have increased the dosage from 12 gm. to 24 gm. of paraaminosalicylic acid daily with only slightly better results.

Tibione appears to be the least effective of the three agents. Since our experience with it is not as great as with paraaminosalicylic acid and streptomycin, final conclusions will not be drawn at this time.

"Primary failures" and "improved" ones following the initial use of paraaminosalicylic acid or tibione can be healed by the use of streptomycin. However, failures that occurred after the initial use of streptomycin were not helped by the subsequent use of either paraaminosalicylic acid or tibione.

The use of streptomycin along with paraaminosalicylic acid may be superior to the use of streptomycin alone (as is generally believed today), but this will be determined only when a significant number of cases in which the two drugs are used can be observed for a sufficient period of time. There is certainly no contraindication to the combined use of paraaminosalicylic acid and streptomycin, and the two drugs are being used in a series of cases in order that this therapeutic combination may be compared with the results obtained from either drug.

SUMMARY

One hundred seventy-seven patients with tuberculosis of the larynx were treated with streptomycin, paraaminosalicylic acid, or tibione as the primary therapeutic agent. Combinations of these drugs were used for the patients in whom the initial agent failed.

The 136 patients treated with streptomycin showed the best results, i. e., the greatest percentage of patients with primary healing, the most rapid healing, and the smallest percentage of reactivations.

The 21 patients treated with paraaminosalicylic acid showed less successful results, while the 20 patients treated with tibione showed the poorest results and the drug appears to be almost useless at this writing.

Healing of a tuberculous larynx usually led to a much-improved general clinical status, despite the absence of change in the roentgen studies of the chest pathology. In 13 cases, healing of the larynx allowed the use of endotracheal anesthesia and thus permitted excisional pulmonary surgery, with its possibility of eradication of the pulmonary lesion.

Clinical studies failed to support the deductions drawn from resistance tests performed solely with the tubercle bacilli found in the sputa. Special studies are being performed on the tubercle bacilli obtained from the several cavities and pleural space after excisional surgery.

Two tuberculous larynges which had been treated with streptomycin were obtained at necropsy. In one case the gross and microscopic pictures revealed the healing by fibrosis, whereas the other case revealed a failure.

The toxic effects of the three agents were evaluated. Paraaminosalicylic acid has no significant toxicity. Streptomycin in doses of 1 gm. a day or less resulted in a very small percentage of characteristic neurotoxic symptoms referable to the vestibular system and practically no hearing loss. However, tibione appears to be more toxic than reported by the German investigators.

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1078 Madison Ave.

Case Reports

SURGICAL INTERRUPTION OF THE ANTERIOR ETHMOID ARTERY IN SEVERE EPISTAXIS

Report of Two Cases

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G. MAIER, M.D.
AND
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NOTWITHSTANDING the empirical fact that about 90% of all nosebleeds will be controlled usually by simple methods, there are a number of cases in which persistent bleeding necessitates the surgical interruption of a major artery.

As the subject of epistaxis has been discussed exhaustively by such authors as Ogura and Senturia,[1] Harkins,[2] and Hallberg,[3] a detailed description of the etiology, treatment, and other aspects of the problem will be omitted here.

The blood supply of the nose is derived from branches of the external carotid artery via the internal maxillary and sphenopalatine vessels, as well as from the internal carotid artery by way of the ophthalmic artery and its branches, the anterior and posterior ethmoidal vessels.

There exist a number of reports of ligations of the common and/or the internal carotid artery, sometimes followed by disastrous results. Publications regarding the ligation of the external carotid artery have appeared less frequently. Spar and Hallberg,[4] for instance, reported 11 cases of such procedures occurring within 18 years at the Mayo Clinic.

Since 1937, when Goodyear [5] performed the ligation of an anterior ethmoidal artery for epistaxis by an approach through the orbit, only 15 instances of this operation have been recorded, as far as we have been able to ascertain from the

From the Veterans Administration Hospital.

Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. Ogura, J. H., and Senturia, B. H.: Epistaxis, Laryngoscope **59**:743-763, 1949.

2. Harkins, H. P.: Hemorrhage in Nose and Throat, A. M. A. Arch. Otolaryng. **55**:8-11, 1952.

3. Hallberg, O. E.: Severe Nosebleed and Its Treatment, J. A. M. A. **148**:355-360, 1952.

4. Spar, A. A., and Hallberg, O. E.: Severe Epistaxis and Its Management: Report of 11 Cases in Which the External Carotid Artery Was Ligated, Ann. Otol. Rhin. & Laryng. **56**:141-151, 1947.

5. Goodyear, H. M.: Nasal Hemorrhage: Ligation of Anterior Ethmoid Artery: Case Report, Laryngoscope **47**:97-99, 1937.

literature at our disposal. To these we wish to add two cases which recently came under our observation, being fully conscious of the likelihood that in other cases this procedure may have been practiced without being recorded.

That rupture of an anterior ethmoidal artery may be the cause of death has been demonstrated at autopsy by Davis.[6] Furthermore, the clinical and anatomical efficiency of the circle of Willis with regard to the ethmoidal arteries must be taken into consideration (Schorstein[7]); i. e., in spite of the ligation of the internal carotid artery, the flow of blood through the ipsilateral ethmoidal arteries may be maintained from the contralateral side through the circle of Willis.

Interruptions of an Anterior Ethmoidal Artery, With or Without Ligation of Other Arteries

No.	Author and Year of Publication	Age	Sex	Etiology	Anterior Ethmoidal and Method	Posterior Ethmoidal and Method	Ligation of External Carotid	Ligation of Internal Carotid	Ligation of Common Carotid
1	Goodyear,[5] 1937	74	M	Undetermined	Primary ligation	.....	.....	.....	.....
2	Windham,[8b] 1945	74	F	Undetermined	Primary ligation	.....	.....	.....	Secondary
3	Windham,[8b] 1945	67	M	Diabetes mellitus	Primary ligation	.....	.....	.....	.....
4	Reading,[8c] 1945	44	..	Undetermined	Secondary ligation	.....	Primary	.....	.....
5	Williams,[8d] 1945	23	M	Trauma	Secondary diathermy	Secondary diathermy	Primary	.....	.....
6	Weddell, Macbeth, Sharp, and Calvert,[8e] 1946; Macbeth, 1948[8f]	11	M	Trauma	Secondary ligation	.....	Primary	.....	.....
7		24	M	Trauma	Quaternary ligation and accessory arteries	.....	Primary	Secondary	Tertiary
8		34	M	Trauma	Secondary ligation	Secondary diathermy	.....	.....	Primary
9		12	M	Trauma	Primary ligation	.....	.....	.....	.....
10		30	M	Telangiectasis	Primary ligation	.....	.....	.....	.....
11		62	M	Hypertension	Primary diathermy and ligation	Primary diathermy	.....	.....	.....
12	Fitz-Hugh and Risher,[8f] 1949	67	M	Hypertension and arteriosclerosis	Secondary ligation	.....	Primary	.....	.....
13	Sackadorf,[8g] 1950	74	M	Arteriosclerosis; Kahn +	Secondary ligation and section	Secondary section	Primary	.....	.....
14	Hallberg,[8] 1952	?	?	?	Primary ligation	.....	.....	.....	.....
15	Morse,[81] 1952	20	M	Polypectomy	Secondary ligation	.....	Primary	.....	.....
16	Authors' cases, 1952	60	M	Hypertension	Secondary ligation	.....	Primary	.....	.....
17		44	F	Hypertension	Secondary diathermy and clip	.....	Primary	.....	.....

The surgical interruption of a major artery usually is undertaken in cases of epistaxis only after other means have failed to control the bleeding. These methods, among others, include the application of various styptics, blood transfusions, the administration of ascorbic acid, vitamin K, thrombin, and calcium, chemical and/or electrical coagulations, and anterior and posterior packings of the nose with different materials.

6. Davis, E. D. D.: Severe Epistaxis, Difficult to Control, Brit. M. J. **1**:721-723, 1939.

7. Schorstein, J.: Carotid Ligation in Saccular Intracranial Aneurysms, Brit. J. Surg. **28**:50-70, 1940.

Of the 17 cases on record (Table [8]), the epistaxis was caused by trauma in 6, including 1 case of severe postoperative epistaxis following a nasal polypectomy. In five cases there were arteriosclerosis and/or hypertension, including one in which there was, in addition, a positive reaction to the Kahn test. In four instances the etiology could not be determined. In one case telangiectasis gravidarum and diabetes mellitus, respectively, were given as the causes of bleeding.

When the bleeding can be observed definitely to originate above the middle turbinate, ligation of the anterior ethmoidal artery alone will stop the hemorrhage. This was the procedure in 5 of the 17 cases. The posterior ethmoidal artery, being usually of small caliber, does not play any significant role in nosebleed. However, it was coagulated as an additional precaution in three cases. In another instance it was sectioned without bleeding.

Most of the interruptions of the anterior ethmoidal artery were preceded by ligation of the external carotid artery (nine cases). In two cases ligation of the common carotid artery was performed previously. The epistaxis in one case necessitated ligation of the external carotid, internal carotid, common carotid, anterior ethmoidal, and accessory ethmoidal arteries, in that order. In one case the primary ligature of the anterior ethmoidal artery had to be followed by ligation of the common carotid artery.

REPORT OF CASES

Case 1.—D. S., a 60-year-old white man, was known to have hypertension of three years' duration and was admitted on March 11, 1952, with a history of severe epistaxis of four days' duration. He stated that three days previous to admission he was seen by a private physician, who used anterior packs and stopped the bleeding for four hours. However, the patient started to bleed again and was referred to this hospital for treatment.

Physical examination on admission revealed a very pale man with a blood pressure of 176/100. There was profuse bleeding from the left nostril, but no definite bleeding point was seen. Hemoglobin averaged 9 gm. per 100 cc., and the red blood cell count averaged 3,300,000 per cubic millimeter.

The patient was treated immediately with anterior and posterior nasal packs; however, he continued to bleed. He was given 1,500 cc. of whole blood, and on March 13 the left external carotid artery was ligated. The patient continued to bleed after operation, and he was given another 3,000 cc. of whole blood from March 13 to 17. On March 17 ligation of the anterior ethmoidal artery was done through a curvilinear incision and after careful elevation of the orbital periosteum. While on the operating table and immediately thereafter, the patient was given 1,000 cc. of whole blood. After ligation of the anterior ethmoidal artery the epistaxis ceased and did not recur. On the seventh postoperative day the patient was discharged and has had no recurrence of bleeding to this date.

8. (*a*) Goodyear.[3] (*b*) Windham, R. E.: Cause and Treatment of Rhinopharyngeal Hemorrhages, Texas J. Med. **40**:484-487, 1945. (*c*) Reading, P.: Ligature of Anterior Ethmoidal Artery for Arrest of Profuse Epistaxis, Brit. M. J. **2**:848-849, 1945. (*d*) Williams, J. L. D.: Ethmoidal Epitaxis Necessitating Operation: Case Report, J. Laryng. & Otol. **60**:292-293, 1945. (*e*) Weddell, G.; Macbeth, R. G.; Sharp, H. S., and Calvert, C. A.: Surgical Treatment of Severe Epistaxis in Relation to the Ethmoidal Arteries, Brit. J. Surg. **33**:387-392, 1946. (*f*) Fitz-Hugh, G. S., and Risher, J. C.: Epistaxis: Report of a Case of Ligation of the External Carotid and Anterior Ethmoid Arteries, Arch. Otolaryng. **49**:564-565, 1949. (*g*) Sackadorf, H.: Epistaxis: Case Report: Report of a Case of Ligation of External Carotid and Anterior Ethmoidal Arteries, and Section of Posterior Ethmoidal Artery, Laryngoscope **60**:1125-1128, 1950. (*h*) Hallberg.[3] (*i*) Morse, L. M.: Epistaxis: Report of 3 Cases, Eye, Ear, Nose & Throat Month. **31**:150-151, 1952. (*j*) Macbeth, R. G.: Anterior Ethmoidal Haemorrhage, J. Laryng. & Otol. **62**:42-47, 1948.

CASE 2.—K. W., a 44-year-old white woman, had been treated for epistaxis by her physician for five months previous to admission. She was known to have suffered from hypertension for 10 years. When admitted to this hospital, on the evening of May 8, 1952, she had been bleeding from her nose for 12 hours. Her blood pressure was 190/110, the red blood cell count was 3,800,000 per cubic millimeter, and hemoglobin was 11.5 gm. per 100 cc.

Examination of the nose revealed severe bleeding from the left nostril, apparently coming from above the posterior third of the lower turbinate. Evidence of cauterization over several areas of the septum on the left side was noted. A posterior-anterior nasal pack immediately was inserted, with cessation of bleeding. The following morning the patient again started to bleed through the packing. Hemoglobin was 10.5 gm. per 100 cc., and the red blood cell count was 3,300,000 per cubic millimeter. Removal of the old packing and insertion of another posterior-anterior nasal pack did not control the bleeding. Ligation of the left external carotid artery between the superior thyroid and the lingual artery then was performed, and a transfusion of 500 cc. of blood was given.

The bleeding ceased but returned again profusely within a few hours. Another transfusion of 500 cc. of blood was given. The left anterior ethmoidal artery was exposed in the orbit through a curvilinear incision and then was coagulated, after application of a silver clip. The patient was discharged after five days of hospitalization without recurrence of bleeding.

COMMENT

Had it been possible to ascertain the origin of the nasal bleeding in these cases, interruption of the anterior ethmoidal artery alone would have been sufficient in controlling the hemorrhage, without a preliminary ligation of the external carotid artery. Surgical interruption of the anterior ethmoidal artery is a comparatively simple technical procedure. It is well known to every surgeon performing the Ferris Smith external ethmoidectomy or an external frontoethmoidosphenoidectomy. In both cases the patients are free from diplopia.

SUMMARY

Two cases of severe epistaxis are reported in which surgical interruption of the anterior ethmoidal artery, after ligation of the external carotid artery, proved to be lifesaving.

Clinical Notes, New Instruments and Techniques

MODIFIED YANKAUER ASPIRATOR

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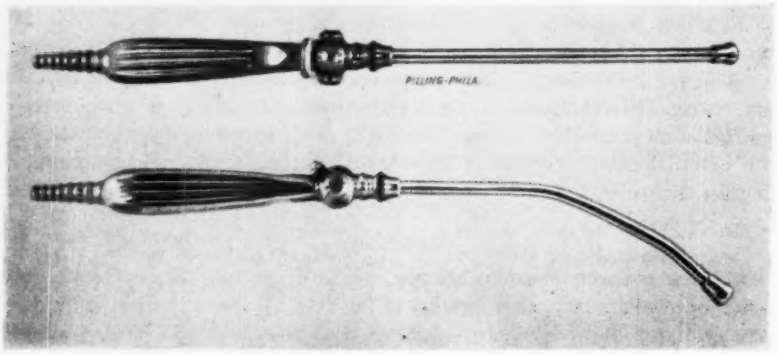
In operating rooms equipped with central suction and its long tubes, or where long tubes are necessary for other reasons, there is a time element lost in "building up" vacuum in the tubes. We have found, for instance, that it takes 14 seconds to build up 20 in. (51 cm.) of vacuum in a closed tube 15 ft. (46 meters) long and an inside diameter of ¼ in. (0.6 cm.) with a suction



Cut-off for Yankauer aspirator.

apparatus capable of producing 20 in. of vacuum. Once established, this vacuum is constant. The shorter the tube, the less time is necessary to build up the vacuum.

The cut-off we devised on the Yankauer aspirator shortens the length of time necessary for building up a vacuum in the usual open-end aspirator and tube. With the cut-off in the "on" position, maximum vacuum is obtained and ready for instant use. I have found it most useful in sudden profuse hemorrhage.

3601 Stokley St.

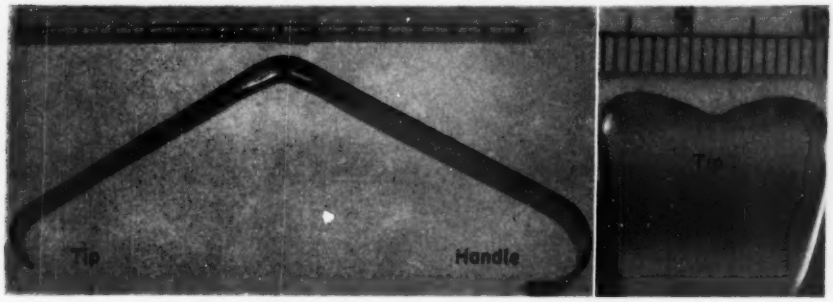
From Otolaryngological Service "B," Episcopal Hospital.
Read before the College of Physicians of Philadelphia, Section of Otolaryngology, Feb. 6, 1951.

NEW SOFT-PALATE RETRACTOR

SHIRLEY HAROLD BARON, M.D.
SAN FRANCISCO

Direct exposure of the nasopharynx for removal of lymphoid-tissue remnants after the main adenoid mass has been extirpated has wide acceptance as the most satisfactory method of insuring an efficient adenoidectomy.

Adequate exposure of the nasopharynx sometimes presents a bit of a problem when a fixed-tongue-blade type of mouth gag, such as the Davis or McIvor, is used. This type of gag gives an excellent view of the oropharynx and hypopharynx but has the disadvantage of fixing the anterior pillars. This diminishes the amount of cephalad retraction that can be exerted upon



A new soft-palate retractor.

the uvula and the soft palate, thus limiting the nasopharyngeal exposure. To improve this exposure, an additional pull forward is required. To accomplish this, a new soft palate retractor has been devised.

The retractor [1] presented has a "butterfly" tip (Figure) with a central notch that prevents the uvula from slipping from side to side. The tip is short enough to avoid the hard palate but long enough for a good "hold" on the soft palate. The handle is modeled after the Love [2] retractor. The instrument is made of brass and will not bend. One size works equally well for children and for adults. It is especially useful in exposing the nasopharynx in those patients who have scarred shortened pillars from previous surgery.

516 Sutter St. (2).

1. The retractor is made by the Storz Instrument Company, St. Louis.

2. Love, A. A.: New Soft Palate Retractor and Adenoid Punch, Arch. Otolaryng. **19**:66-67 (Jan.) 1934.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF OCT. 1, 1952

Audicon Models 400 & 415
Mfr., National Earphone Co., Inc.
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Mfr., Audio Company of America
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Phoenix, Ariz.

Audivox Model Super 67
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Symphonette Model

Beltone Mono-Pac Model M
Mfr., Beltone Hearing Aid Co.
1450 W. 19th St.
Chicago 8

Cleartone Model 500

Cleartone Model 700

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2
Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Dysonic Model 1
Mfr., Dynamic Hearing Aids, Inc.
149 Church St.
New York 7

Electrocar Model C
Mfr., American Earphone Co., Inc.
10 E. 43rd St.
New York 17

Gem Model V-35

Gem Model V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69 & 97
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corporation
708 W. 40th St.
Minneapolis 8

Maico UE Atomeer

Maico Quiet Ear Models G & H

Maico Model J
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal & Magnetic) Aurophone Model 200

1947-Mears Aurophone Model 98
Mfr., Mears Radio Hearing Device Corporation
1 W. 34th St.
New York 1

Micronic Model 303

Micronic Model "Mercury"

Micronic Star Model
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Audiomatic T-5

Microtone Classic Model T-9

Microtone Model T-10

Microtone Model T612

Microtone Model 45

Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506

Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Otarion Model E-4

Otarion Models F-1, F-2 & F-3

Otarion Models G-2 & G-3

Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D "Top-Twin-Tone"

Paravox Model J (Tiny-Myte)

Paravox Model XTS ('Xtra-Thin)

Paravox Model Y (YM, YC & YC-7) [Veri-Small]

Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Permo-Magnetic (Multipower)

Radioear Permo-Magnetic (Uniphone)

Radioear All-Magnetic Model 55

Radioear Model 62 Starlet

Radioear Model 72

Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mount Lebanon
Pittsburgh 16

Distr., Radioear Corporation

Rochester Model R-1

Rochester Model R-2

Mfr., Rochester Acoustical Laboratories, Inc.
117 Fourth St., S. W.
Rochester, Minn.

Silvertone Model J-92

Silvertone Model P-15

Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck and Co.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99

Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Model 700

Sonotone Model 900

Sonotone Models 910 & 920

Sonotone Model 925

Sonotone Model 940

Sonotone Model 966

Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E

Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Model 97

Telex Model 99

Telex Model 200

Telex Model 300-B

Telex Model 400

Telex Model 500

Telex Model 1700

Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50

Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Model Royal

Tonemaster Cameo Model

Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Model 95

Unex Midget Model 110

Unex Models 200 & 230

Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2

Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 & 66

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Zenith Model 75

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)

Mfr., A. N. Brooks Company
684 S. Bonnie Brae St.
Los Angeles 5

Aurex Semi-Portable

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid

Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50

Mfr., Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

AN EXPERIMENTAL STUDY OF AUDITORY DAMAGE FOLLOWING BLOWS TO THE HEAD. HAROLD F. SCHUKNECHT, WILLIAM D. NEFF, and HENRY B. PERLMAN, Ann. Otol. Rhin. & Laryng. **60**:274 (June) 1951.

Schuknecht, Neff, and Perlman subjected 10 conditioned cats to head blows in an effort to analyze the relationship of inner ear pathology to the resultant deafness in the animals. Post-concussion audiometry demonstrated high-tone losses, particularly around 4000 cps. Significant middle ear and brain damage could not be detected on gross or microscopic study.

The primary injury was found to be a degenerative change in the organ of Corti. The upper portion of the basal coil of the cochlea was most susceptible to injury. The stages of end-organ damage noted in the order of severity were the following: (1) loss of the external hair cells; (2) loss of both external and internal hair cells; (3) flattening of the organ of Corti, and (4) complete disappearance of the organ of Corti. Secondary degeneration of nerve fibers and ganglion cells supplying the damaged regions of the cochlea was found. Blood cells remaining in the cochlea for as long as four months appeared to have set up no reaction.

It is concluded that a blow to the head creates a pressure wave in the skull which is transmitted to the cochlea. Violent displacement of the basilar membrane is thereby produced, causing injury to the organ of Corti.

STEELE, Kansas City, Kan.

OTOSCLEROSIS OF THE OSSEOUS HORIZONTAL SEMICIRCULAR CANAL. EWING SELIGMAN and GEORGE E. SHAMBAUGH, Ann. Otol. Rhin. & Laryng. **60**:375 (June) 1951.

Seligman and Shambaugh using the binocular operating microscope and a continuous flow of sterile isotonic sodium or chloride solution or Ringer solution to keep the fenestration field clear of debris have found otosclerosis of the osseous horizontal semicircular canal on occasion. It appears to be seen more frequently in patients with some cochlear nerve involvement, as found on preoperative testing, and suggests that the more extensive the otosclerotic bone lesion the likelier is the nerve to degenerate. Accordingly, the prognosis is poorer.

It has been observed that the endosteal membrane is an effective limiting membrane to osteogenic encroachment on the lumen of the canal. Therefore, hemming the fenestra with endosteum has seemingly aided in the prevention of osseous closure of the window. In this technique the endosteal membrane from the mouth of the fenestra is folded over the sharp, knife-like edge of the completed fenestra, so as to insure contact with the periosteum of the flap and subsequent primary healing of the cut bone edge.

STEELE, Kansas City, Kan.

SURGERY IN MÉNIÈRE'S DISEASE: A NEW OPERATION WHICH PRESERVES THE LABYRINTH. SAMUEL ROSEN, Ann. Otol. Rhin. & Laryng. **60**:657 (Sept.) 1951.

Rosen has treated 14 patients who had labyrinthine hydrops by transtympanic section of the chorda tympani nerve or both the chorda tympani nerve and the tympani plexus.

Eleven patients reportedly have had no vertigo following operation, while in two the vertigo has persisted. Tinnitus disappeared altogether in one patient, diminished to a bearable level in seven, and remained unaffected in six patients. Hearing was not destroyed in any instance.

According to Rosen, the procedure is simple, requiring only two or three days' hospitalization, is performed with local anesthesia, and can be safely carried out on both ears in cases of bilateral Ménière's disease. It should be tried before resorting to radical destructive labyrinthotomy.

STEELE, Kansas City, Kan.

ON THE FORMATION OF OTOLITHS. THURE VILSTRUP, Ann. Otol. Rhin. & Laryng. **60**:974 (Dec.) 1951.

The otolithic granules in the shark labyrinth appear to be of endogenous and exogenous origin. The endogenous granules are produced above the macula in the early stages of development or elsewhere postnatally, while the exogenous granules are introduced into the labyrinth from external sources after birth. These are situated above the macula.

Endogenous otoliths, making up about 90% of the total otolithic mass, are formed either at the site of the macula or in the epithelial lining of the endolymphatic duct. In the latter site they have the appearance of vacuoles containing calcium carbonate. They migrate out of, or are discharged from, the epithelium into the lumen of the duct. They then pass down to the macular gelatinous substance in which they become imbedded. This procedure takes place after the birth of the animal.

Exogenous otoliths enter the endolymphatic duct through its opening on the cranium of the shark and find their way to the macular gelatinous substance. Analysis proves them to be grains of sand (quartz).

STEELE, Kansas City, Kan.

CORRELATION OF THE PREDICTED WITH THE ACTUAL RESULT OF FENESTRATION IN 164 CONSECUTIVE CASES. GEORGE E. SHAMBAUGH JR., Laryngoscope **62**:461 (May) 1952.

There is a way to predict the gain in hearing to be expected from a fenestration operation. That bone-conduction audiograms are accurate if done properly and with certain corrections has been shown by Carhart and Hayes. Bone-conduction audiograms must be taken in this determination. Carhart has shown that before there is a lowering of bone conduction (average 10 db. for 512, 1024, and 2048 dv.) in otosclerosis. This is called the otosclerotic notch. The residue of unrestored conductive loss after fenestration is 25 db. (because of the action of normal lever action of the drum membrane and mobile ossicles). If one therefore subtracts 25 db. from the bone-conduction level at 512, 1024, and 2048 and adds 10 db. for the notch correction or by combining the above figures merely subtracts 15 db. from the bone-conduction level, the proper prediction of the postfenestration hearing will be obtained.

One hundred and sixty-four cases are reported. It is noted that the greater the suitability of the case for fenestration the more accurate will be the prediction gain.

HITSCHLER, Philadelphia.

THE PROBLEM OF THE PRESCHOOL DEAF CHILD. JOHN E. BORDLEY, Laryngoscope **62**:514 (May) 1952.

In order to determine the extent, cause, and kind of hearing loss in the preschool child, there must be the proper equipment and also sufficient cooperation from the parents. During any hearing-speech training program the doctor should be in continual touch with the patient in the role of counselor.

Diagnosis is difficult. A most careful history is necessary. A printed questionnaire (shown) is helpful. Examination must include that of the nose, pharynx, and Eustachian orifices, as well as of the ears and hearing. General performance tests aid in determining the child's mental state. The hearing itself is tested by the tuning forks, by monitored sound effects in a sound-proof room, and by the audiometer. The latter includes both the psychogalvanic skin-resistance audiometer and the pure-tone subjective audiometer (when possible).

An analysis of the etiologic factors involved in 485 cases is given. Two causes are emphasized: mothers receiving immune globulin during the first trimester of their pregnancy and cyanosis at birth.

Early diagnosis, with an intelligent training program using amplification, is most important and may mean the difference between a normal and an abnormal upbringing.

HITSCHLER, Philadelphia.

OCCUPATIONAL DEAFNESS. VASSALO DE MUMBERT, Bol. Españ. otorrinolaring y broncoesofagol. **5**:19, 1952.

In a comprehensive review replete with many references Mumbert presents a splendid discussion of occupational deafness. He divides the problem into (*a*) acoustic trauma, (*b*) barometric trauma, and (*c*) toxic trauma.

Under the heading of acoustic trauma he includes all forms of occupations associated with noises, either persistent or intermittent, of varying intensity and varying pitch. He stresses the difficulty of diagnosis, and with the patient under treatment he suggests the removal of the cause. However, realizing that economic problems will present barriers to this problem, he suggests that state or federal legislation be instituted similar to that employed for other forms of occupational hazards. Among his suggestions are the use of cotton or other forms of ear stoppers, the attempt to eliminate many vibrations in the various industrial plants, the interruption of work without any noise for varying periods throughout the day, the audiometric control of every worker admitted to the industry, the inclusion of a preemployment auditory test followed by examinations every month or during vacations or during sickness, and, lastly, a complete otorhinologic examination prior to occupation with the thought in mind to eliminate any condition that may predispose to hearing loss. In addition to the above recommendations, he advises the administration of large and frequent doses of pyridoxine, since it is his belief that the pathology is essentially centered in the organ of Corti.

His second group includes those cases of deafness sustained in war or in aviation. Many of the cases in this group are so frequently associated with underlying local conditions that it is extremely important to evaluate these factors. Among these are local infections of the nose and throat; anatomic factors, such as hypertrophy of the adenoids, deviated septa, etc.; traumatic causes, such as local cicatrization in the region of the Eustachian tube orifice; static causes, such as malocclusion of the mandible; allergic causes, and finally, a mechanical obstruction of the Eustachian tube by benign or malignant tumors of the cavity or by hypertrophy of the posterior ends of the inferior turbinates. Attention to the above causes is extremely important. With the patient under therapy Mumbert further suggests the use of cotton in the ears, oxygen rooms in airplanes, and chewing of gum during airplane flights, particularly during descent. In addition, here, too, he recommends massive doses of pyridoxine.

In his last group he includes all those cases of occupational hazards associated with various forms of poisoning, such as lead, arsenic, benzene, and trinitrotoluene. He discusses the symptomatology of each particular type of toxication and suggests change of occupation plus elimination of the poison, where possible. Large doses of pyridoxine and potassium iodide and supportive treatment are indicated.

PERSKY, Philadelphia.

PROSTHESIS TREATMENT AND ITS HEALING EFFECT ON CENTRAL PERFORATIONS OF THE TYMPANIC MEMBRANE. AGNAR HALL, Acta oto-laryng. **39**:136 (June) 1951.

A method for prosthesis treatment of central perforations with 5% celluloid solution in acetone is described. If the perforation exceeds 2.5 mm. in diameter, the perforation is first covered with a 9 mm. round plate of Yoshino paper. Results were good on old, as well as new, perforations. Results could not be expected unless the mucous membrane of the middle ear was normal. In instances of atrophic and scarred mucous membrane the healing does not result. To get a fully satisfactory closure of the perforations, the prosthesis must be able to maintain its continued integrity, be waterproof, and be resistant to physiological pressures. In successful cases the prosthesis aids hearing and in some instances dizziness and tinnitus.

ROBERT LEWY, Chicago.

Larynx

THE PRESENT STATUS OF TRACHEOTOMY IN BULBAR POLIOMYELITIS. ROBERT E. PRIEST, LAWRENCE R. BOIES, NEILL F. GOLTZ, L. IAN YOUNGER, and ROBERT L. KOLLER, Ann. Otol. Rhin. & Laryng. **60**:468 (June) 1951.

According to Priest and co-authors, 26 papers on the subject of tracheotomy in cases of bulbar poliomyelitis have been published since 1946. These are reviewed and discussed. While

varying opinions are expressed in these writings, most of the authors favor tracheotomy in certain instances. Secretional obstruction to the airway is recognized as a prime indication, but pharyngeal paralysis alone is thought not to be a proper indication for the operation. Inability on the part of the patient to clear his own airway of secretions, pharyngeal distortion resulting from paralysis, and mechanical obstruction are cited as additional indications.

Some authors advise against tracheotomy on the grounds that the operation is dangerous and mutilating. Others believe that its early utilization prevents some brain damage by assuring more adequate oxygen supply during the course of the disease.

The authors state their position based on their own experience, as well as on that of others as follows, in summary: 1. Tracheotomy should be done early when needed. 2. Done properly, it adds little risk to the patient. 3. The mutilating nature of the procedure has been overexaggerated. 4. Tracheotomy contributes greatly to prognosis when the patient cannot clear his own airway. 5. Transtubal humidified oxygen under pressure is an important form of adjuvant therapy. 6. There are no hard and fast indications for tracheotomy. 7. The operation should be done only if other methods of clearing the airway of obstructive secretions have failed. 8. Tracheotomy has a limited, but important, place in the treatment of bulbar poliomyelitis.

STEELE, Kansas City, Kan.

BRONCHOGRAPHY IN THE INFANT AND VERY YOUNG CHILD. FERNAND G. EEMAN, Ann. Otol. Rhin. & Laryng. **60**:793 (Sept.) 1951.

The history of bronchography is reviewed, going back to 1914 when Weingartner accidentally introduced bismuth into the tracheobronchial tree.

The following reasons for the unpopularity of the use of bronchography in children are presented and commented upon, as follows:

1. The Risk of Anesthesia: Utilization of tetracaine (pontocaine®) hydrochloride by fine spray or nebulizaton is advocated. Slow administration (20 to 30 minutes) and high dilution of the agent employed are stressed. Eeman finds proof lacking of increased sensitivity of children to topical anesthetic drugs as compared to adults. Very young children and infants may have satisfactory examinations without anesthesia.

2. The Risk of the Technical Procedure of the Introduction of the Opaque Oil: Use of a soft catheter, introduced transorally or transnasally, obviates the necessity for bronchoscopy except in a few cases. [In the reviewer's opinion, if bronchography is indicated, so also is preliminary bronchoscopy.]

3. The Risk of Respiratory Trouble Due to the Filling of the Tracheobronchial Tree: The smaller capacity of the infant tracheobronchial tree should be remembered, for overfilling is as dangerous in infants as in adults. Fluoroscopic control is of value.

4. The Risk of Intoxication Due to the Iodine: This risk may be avoided by observing precautions in known cases of acute tuberculosis; by using iodine skin tests two days prior to bronchography; by not exceeding the necessary amount of radiopaque oil; by utilizing topical rather than general anesthesia, and by aspirating the iodized oil immediately following bronchography or instituting postural drainage.

STEELE, Kansas City, Kan.

AQUEOUS CONTRAST MEDIA IN BRONCHOGRAPHY. CHARLES M. NORRIS and HERBERT M. STAUFFER, Ann. Otol. Rhin. & Laryng. **60**:802 (Sept.) 1951.

Observations on the properties and uses of aqueous contrast media for bronchoscopy are recorded. Norris and Stauffer studied "umbradil viscous B" (product of A. B. Astra, Södertälje, Sweden) and "ioduron B" (product of Cilag, Ltd., Schaffhouse, Switzerland) in comparison to the iodized oils in common use in the United States.

The advantages of the aqueous iodine media are more rapid elimination of the contrast substance (iodopyracet), chemical stability, viscosity which may be varied at will, and freedom from systemic toxic effect. Although unproved now, local tissue reaction is probably of less degree and shorter duration.

The disadvantages of the aqueous contrast media are greater irritant effect on the tracheobronchial tree and lack of more informative bronchograms than those secured with iodized oil.

Details of technique are described. The literature describing effects of oily and aqueous preparations for bronchography on the lung parenchyma is reviewed.

STEELE, Kansas City, Kan.

CARCINOMA OF THE LARYNX. JULIUS W. MCCALL and WILLIAM R. FISHER, Laryngoscope **62**:475 (May) 1952.

This is a review of 194 cases of carcinoma of the larynx. The symptoms, treatment, and results are discussed and tabulated. The importance of hoarseness is noted. In 70% of the cases in which there was a five-year survival hoarseness was the only symptom.

HITSCHLER, Philadelphia.

Nose

INFERIOR MEATAL ACCESSORY OSTIA: REPORT OF A CASE. ADRIAN J. DELANEY and HARRY R. MORSE, Ann. Otol. Rhin. & Laryng. **60**:635 (Sept.) 1951.

Delaney and Morse report a case of bilateral inferior meatal accessory antral ostia involving a 24-year-old white woman, apparently with a congenital basis. Nasopharyngoscopy and roentgenography revealed normal maxillary sinuses. Middle meatal ostia were found in the usual location bilaterally. While such an anomaly is difficult to explain embryologically, the absence of any evidence of an acquired condition causes Delaney and Morse to assume a congenital etiology. Reportedly, no other such case has been recorded in the medical literature.

STEELE, Kansas City, Kan.

INTRANASAL ENCEPHALOCELE. W. P. WILLIAMSON and P. A. BARELLI, J. Neurosurg. **8**:231, 1951.

Encephalocele, or congenital protrusion of the brain or meninges through the skull, is not uncommon. The most frequent site of such a lesion is occipital. In a series of 84 reported encephaloceles, 63 were occipital, 9 parietal, 6 frontal, 5 nasal, and 1 presenting through a defect in the cribriform plate into the nasopharynx.

Williamson and Barelli report two cases of intranasal encephalocele with relief of rhinorrhea by intracranial approach. In the first case, the broad nose, the history of two attempts at removal of a nasal polyp, and the long history of rhinorrhea in the absence of head trauma made the diagnosis obvious. In the second case, since there was a history of a linear skull fracture two years before, the surgeon assumed that the rhinorrhea was simply traumatic and had not anticipated the true nature of the lesion until its actual exposure at operation. The skull fracture had undoubtedly torn the base of the encephalocele to precipitate the initial onset of the spinal-fluid leakage.

Williamson and Barelli point out that intranasal encephalocele is probably commoner than is generally believed and that it should be considered in the differential diagnosis of nasal polyp and rhinorrhea.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.]

BACTERIOLOGIC AID IN RHINOLOGIC DIAGNOSIS AND TREATMENT, IMPROVED CRITERIA. JOSEPH L. GOLDMAN, Laryngoscope **62**:406 (April) 1952.

Comparatively simple bacteriologic investigations can help greatly in the diagnosis and management of nasal and sinus disease. Goldman feels that often one cannot depend upon the amount or gross appearance of the secretions to indicate the amount of infection. Microscopic examination and culture must be made. One must demonstrate the presence of organisms to prove the presence of infection. Similar studies are necessary to demonstrate allergic rhinitis. In general, there are pathogenic organisms, polymorphonuclears, and little or no mucus in infections, while in allergic conditions there are eosinophiles (usually), mucus, and no (pathogenic) organisms. It must be noted that potential pathogens may be found in normal noses, and occasionally a so-called nonpathogenic organism may cause a definite sinusitis. Repeated cultures must be made to substantiate the latter.

Tests of the susceptibility of the different organisms to the various antibiotics aid in treatment.

HITSCHLER, Philadelphia.

THE TREATMENT OF NASAL POLYP BY THE RUSKIN METHOD. JIMENEZ CERVANTES, Laryngoscope **62**:535 (May) 1952.

The patients in eight cases of nasal polyposis were treated as Rushkin suggested, namely, by the instillation of procaine penicillin into the maxillary sinus. There was no improvement in any case.

HITSCHLER, Philadelphia.

Miscellaneous

USE OF SERIAL ANGIOGRAPHY IN DIAGNOSIS OF SPACE OCCUPYING INTRACRANIAL LESIONS. H. C. VORIS, Am. J. Roentgenol. **67**:360, 1952.

Voris reports briefly 11 cases in which serial cerebral angiography was performed. In most of the cases the percutaneous method was used, with 35% iodopyracet as the medium. Voris' routine in most cases was to take a series of six films during a period of 4½ seconds in both the anteroposterior and the lateral projection. For each series of six films 15 cc. of iodopyracet was used. Preparation for the percutaneous injection consisted of a test for sensitivity to iodopyracet and the administration of 0.3 gm. of pentobarbital sodium from 60 to 90 minutes before the procedure.

The article illustrates the various types of displacement of the cerebral blood vessels by space-occupying intracranial lesions. The necessity of both anteroposterior and lateral films is emphasized. Voris points out that the demonstration of the arterial supply and the venous drainage of vascular neoplasms of the brain is of great aid in planning the surgical procedure.

WEILAND, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.]

CHILDHOOD PNEUMONIA, A COMMON CAUSE OF BRONCHOPULMONARY DISEASE. WALTER FINKE, A. M. A. Am. J. Dis. Child. **83**:755 (June) 1952.

Finke thinks that bronchopulmonary diseases are a frequent aftermath of childhood pneumonia. The bronchoscopist would like to know if this is responsible for bronchiectasis and other conditions calling for direct tube exploration. Finke states that superficial antibacterial treatment given at the onset of respiratory illness may alter the whole picture and make "differentiation" difficult. Pulmonary resolution may be delayed for weeks or months, and relapses are likely to occur. Acute pneumonia may combine with an old pulmonary condition, such as chronic cough. "Children who experience recurrent tonsillitis, laryngitis, tracheobronchitis, and eventually pneumonia usually show this tendency from early childhood on and not only after entering school. Consequently, the onset of chronic respiratory disease, including bronchiectasis, often can be traced to infancy." Therefore prolonged antibiotic therapy should be carried out after all apparent symptoms and signs have vanished.

This essay was read before the Section on Pediatrics at the 100th Annual Session of the American Medical Association, Atlantic City, June 15, 1951. A prolonged discussion was undertaken by Dr. Camille D. Cayley of New York who stated the following: "Unresolved pneumonia is becoming a myth. Only 3 cases were seen in 1950, against over 20 in 1938." Dr. Cayley took issue with Finke's dissertation. The termination of therapy is determined by signs, symptoms, temperature, roentgenograms, blood count, sedimentation rate, etc. A well-treated patient with pneumonia is usually completely convalescent in 10 to 14 days after hospital care. It may be that patients with virus pneumonia take a little more time to recover. But allergic conditions may precede or follow any incidental disease of the airways. This possibility must always be considered in so-called chronics.

I. W. VOORHEES, New York.

ADENOMA OF THE BRONCHUS. EELCO HUIZINGA and J. IWEMA, Ann. Otol. Rhin. & Laryng. **60**:290 (June) 1951.

This article is a report of 17 cases of adenoma of the bronchus, as well as two cases of cylindroma and one case of adenoma of the trachea, observed at Groningen University Clinic, Groningen, Netherlands.

Adenoma of the bronchus was found to predominate in women and young adults, corresponding to the experience of other investigators. Huizinga and Iwema believe that these tumors originate in the seromucous glands of the bronchial tree and are closely comparable to tumors of the salivary glands, such as mixed tumors. This belief is based on clinical and anatomicopathologic evidence, which is discussed fully.

The therapeutic measures employed in a number of the cases are outlined.

STEELE, Kansas City, Kan.

HISTOLOGIC INVESTIGATIONS IN CHRONIC STREPTOMYCIN POISONING IN GUINEA PIGS. ERNA CHRISTENSEN, HELGE HERTZ, NIELS RISKAER, and GUSTAV VRA-JENSEN, Ann. Otol. Rhin. & Laryng. **60**:343 (June) 1951.

Christensen and co-authors report histologic investigations of guinea pigs treated with streptomycin in daily doses of from 100 to 400 mg. per kilo of body weight.

Corresponding to the clinical effect on the animals, degeneration was found in the nerve cells in the central vestibular and cochlear nuclei. Degeneration of cells in the vestibular ganglion and involvement of the Purkinje cells were found only in the most severely intoxicated animals. The sensory cells of the labyrinth showed no signs of degeneration.

STEELE, Kansas City, Kan.

THE MECHANISM OF DEGLUTITION (SECOND STAGE) AS REVEALED BY CINE-RADIOGRAPHY. J. B. DEC. M. SAUNDERS, COOPER DAVIS, and EARL MILLER, Ann. Otol. Rhin. & Laryng. **60**:897 (Dec.) 1951.

Saunders and co-authors utilized high-speed cine-roentgenography to study the second stage of deglutition. A 35-mm. motion picture camera equipped with an f/0.85 lens was used to photograph the fluoroscopic screen directly during the swallowing of barium. Linograph film, which is very sensitive to green light, was found to be the most satisfactory. Synchronization of the camera shutter with the fluoroscopic tube at 30 frames per second was accomplished by means of a ⅛-HP electric motor attached to a commutator, to allow interruption of current to the tube.

The initial sign of the onset of the second stage of normal deglutition is the ascent of the hyoid bone and the larynx, the lingual muscles having already rolled the food bolus to a point just anterior to the fauces. The base of the tongue is then thrust up and back by the action of the intrinsic muscles, i. e., the mylohyoid, the palatoglossus, and the styloglossus muscles. This up and back motion of the tongue is responsible for the initial movement of the epiglottis from a vertical to a dorsal position, so that the valleculae are opened widely. This motion is transmitted to the epiglottis by contraction of the stylohyoid and the hyoglossus muscles through the hyoepiglottic ligament. It is suggested that the digastric muscles are responsible for the elevation of the hyoid, the arrangement of the intermediate tendon permitting the upward and backward movement of the hyoid, the larynx, and the tongue on later contraction of the stylohyoid and styloglossus muscle.

As food is projected beyond the fauces by the quick (1/20 second) piston-like action of the muscles of the base of the tongue, the soft palate is elevated by action of the levator and tensor veli palatini muscles. Contraction of the palatopharyngeus muscle establishes Passavant's ridge on the postpharyngeal wall. Regurgitation of food into the nasopharynx is prevented by the described muscular action, in addition to the continued upward and backward thrust of the tongue.

While it appears true that liquids may be projected as far as the esophageal opening without action of the constrictor muscles, it is certain that semifluid and solid-food boluses are propelled largely thruogh action of these muscles. Cine-roentgenographic studies in cases of unilateral and bilateral pharyngeal paralysis leave little doubt of the importance of the pharyngeal muscles in the swallowing act.

Action of the epiglottis as a trap door over the laryngeal aperture is proved by high-speed cine-roentgenography. The epiglottis is noted to move from a nearly vertical to a horizontal position when the food bolus is propelled to the valleculae and starts to overflow laterally into the pyriform sinus. The final tipping of the epiglottis downward is quite rapid and delivers the food to the cricoesophageal junction.

Descent of the epiglottis is closely related to the sphincteric closure of the larynx. The aryepiglottic folds are observed to shorten, owing to the contraction of the aryepiglottic and thyroepiglottic muscles, which pull the epiglottis back and down. At the same time, further elevation of the larynx passively tilts the epiglottis by forcing the arytenoid eminences against its base. Closure of the vestibule and approximation of the cords are doubtless due to contraction of the thyroarytenoid, the vocal, and related muscles. This constrictor action is so vigorous that it effectively prevents entrance of food into the larynx in cases of excision of the epiglottis.

Return of the epiglottis to its initial erect position is rapidly accomplished by its elastic recoil, by the action of the hyoepiglottic ligament, and by the anterior excursion of the tongue. The larynx finally returns to its resting position.

Saunders and co-authors present summaries of the available historical notes on deglutition, as well as their own selected cine-roentgenograms of the second stage of the swallowing act.

STEELE, Kansas City, Kan.

THE ALLERGIC FACTOR IN CHRONIC BRONCHOPULMONARY INFECTION. F. W. DAVISON, Ann. Otol. Rhin. & Laryng. **60**:948 (Dec.) 1951.

As indicated by Kahn, Davison states that allergy is probably an expression of hyperimmunity and is the result of an overaction of a physiological mechanism. In an allergic person immunity develops more readily, and with smaller quantities of an antigen, than in a normal person.

In the treatment of asthmatic patients cortisone reduces the physiological overaction to infection, thereby decreasing mucosal edema and submucosal leucocytic infiltration, thus widening the bronchial lumen, relieving the dyspnea, and permitting better drainage. Untreated, such a condition leads to atelectasis and later bronchiectasis. There is, therefore, a need for early and adequate attention to the allergic feature plus antibiotic therapy and bronchoscopic aspiration.

Davison studied 25 cases of asthma in which bronchoscopy was recommended for diagnosis or treatment. Twenty of the patients were over 40 years of age. All the patients had chronic suppurative bronchitis, and four, in addition, had bronchiectasis. In each of the latter there was a clear-cut history of pneumonitis or pneumonia. It would appear, therefore, that bronchiectasis can be prevented if acute bronchopulmonary infections in allergic persons can be promptly and adequately treated with bactericidal doses of the proper antibiotic medication. Cortisone is helpful in reducing the excessive allergic reaction which causes bronchial obstruction and retention of exudate.

Davison's recommended formula for treatment is the intramuscular administration of 800,000 units of procaine penicillin every 4 hours, intramuscular injection of 0.5 gm. of streptomycin every 12 hours, and oral administration of 25 mg. of cortisone every 4 hours. Topical use of antibiotics is not advisable, owing to the likelihood of inducing sensitization. The other common antiasthmatic measures are also employed.

Bronchoscopy is performed either for diagnostic purposes or for aspiration of viscid exudate which cannot be coughed out. In 16 (64%) of the 25 cases there was improvement after removal of the gummy endobronchial exudate. This was most noticeable in the cases of Loeffler's syndrome (allergic pulmonary consolidation).

STEELE, Kansas City, Kan.

ASPIRATING BRONCHOSCOPE FOR THE GENERAL SURGEON, OBSTETRICIAN AND ANESTHETIST. E. B. EMERSON JR., J. A. M. A. **147**:1238 (Nov. 24) 1951.

Emerson presents an emergency aspirating bronchoscope suitable for use by surgeons, obstetricians, and anesthetists. There are several figures with instructions for emergency aspiration of foreign material, such as mucus, blood, and vomitus, in atelectatic lungs of traumatic or postoperative origin or for the removal of aspirated material from the tracheobronchial tree of the flooded lungs of a severely ill patient.

These measures are outlined only because they would be lifesaving in the absence of a trained bronchoscopist and because they have been helpful during Emerson's 3½ years of service on active duty in the armed forces.

GORDON, Philadelphia.

THE FUTURE OF OTOLARYNGOLOGY. EDITORIAL, J. A. M. A. **148**:1508 (April 26) 1952.

Once again there is discussion that the great specialty of otolaryngology is shrinking and that the outlook is dim. These thoughts were brought out by a questionnaire sent out by Lierle.

Another study points out that the specialty has brighter prospects. The addition of reconstructive surgery of the head and neck, the thyroid gland, bronchoesophagology, allergy, audiology, and physical medicine to the study of conventional diseases of the ear, nose, and throat would broaden the specialty and prevent its limitation.

These questions will have to be discussed objectively, calmly, and deliberately, and in the proper places.

OTORHINOLOGIC SURGICAL PROBLEMS IN THE BIOCHEMICAL ERA. SAMUEL F. KELLEY, J. Internat. Coll. Surgeons **17**:240 (Feb.) 1952.

Kelley points out that, though antibiotic therapy has reduced the incidence of otolaryngological infections that required urgent surgery or eventual surgical intervention, it has left these same surgical problems with us and, at the same time has brought about new clinical entities, latent mastoid infections, and irreversible changes in anatomic structures, resulting in impairment of hearing and even total deafness. The latter in part might be considered due to the ineffectiveness of the antibiotics.

Antibiotic therapy alone may be ineffectual in many instances in which the patients would be helped by surgical measures.

Valuable as the antibiotics may be, one must remember that they have given rise to new clinical entities in mastoid infections, which require recognition and may necessitate mastoid operations. Though extremely helpful, antibiotic therapy has not replaced the principles of good surgery. Adequate drainage and thorough eradication of irreversible pathologic conditions remain the keynote in the management of infections requiring surgery.

GORDON, Philadelphia.

A TEN-YEAR SURVEY OF SPEECH DISORDERS AMONG UNIVERSITY STUDENTS. D. E. MORLEY, J. Speech & Hearing Disorders **17**:25 (March) 1952.

A total of 33,339 students at the University of Michigan have received speech examinations during the past 10 years. The incidence of clinically defective speech was 3.85%. A percentage of 50.70 had articulation defects, 25.48% were stutterers, 15.04% had voice defects, and 8.75% had miscellaneous defects, such as cleft palate and foreign accent. The number of speech defectives detected during the 10-year period has decreased. More men have speech defects than women.

PALMER, Wichita, Kan.

MEASUREMENT OF THE INTELLIGENCE AND PERSONALITY OF THE DEAF: A REVIEW OF THE LITERATURE. STANLEY BERLINSKY, J. Speech & Hearing Disorders **17**:39 (March) 1952.

This article is a review of the literature concerning the intelligence and personality of the deaf. Psychological studies of the intelligence and personality of the deaf indicate that the two factors are not entirely independent. Good adjustment on the part of a person tends to be associated with his intelligence. The instruments used to describe the intelligence and personalities of the deaf do contribute effectively to a broader knowledge of the deaf.

PALMER, Wichita, Kan.

HORMONES AND OTOLARYNGOLOGY. JOHN J. MAHONEY, Laryngoscope **62**:160 (Feb.) 1952.

This presentation deals with the physiology of the endocrine glands and their importance to medicine generally and to otolaryngology in particular. A hormone is a chemical entity produced by a gland. It circulates throughout the whole body; hence it may act on various organs or tissues. There are interrelations of balance and antagonism among the various hormones. Administration of a hormone depresses the action of the body gland which produces that hormone. With these general principles in mind the actions of the various glands are

described. It is noted that there is rejuvenation of the nasal membranes from a nonciliated to a ciliated type in elderly women given estrogen therapy. Also, there are cases of asthma resulting from allergy to endogenous estrone and cases of chronic migraine resulting from allergy to endogenous progesterone.

HITSCHLER, Philadelphia.

THE PRESENT STATUS OF GLOBUS HYSTERICUS. RALPH G. RIGBY, Laryngoscope **62**:401 (April) 1952.

Globus hystericus is a fairly common condition. It is likely to be overlooked or disregarded by the physician. The condition may be of psychic or organic origin. It should be noted that the hysterical patient wants to be noticed without interference in his personal habits. An outstanding feature of a hysterical patient is his apparent emotional placidity and general air of detachment from his surroundings. He is interested in his symptoms, but his interest is rather that of an onlooker than of a participant.

Organic causes of this symptom may have to do with infections of the pharynx or the upper esophagus, gastrointestinal or thyroid gland disorders, tumors of the vocal cords, allergies, thyroglossal-duct cysts, a marked hypertrophy of the lingual tonsil, or marked lipping of the cervical vertebra, with the so-called inflammatory reaction in the soft tissues anterior to the lipping.

Treatment depends on elimination of the organic cause, if present, and, if of a psychic origin, it depends on a good relationship between the patient and doctor, so that the basis of the psychosis can be determined and, if possible, eliminated.

HITSCHLER, Philadelphia.

ACTH AND CORTISONE IN OTOLARYNGOLOGY. DAVALOS L. HECTOR, An. Soc. Mex. Otorinolaring. **3**:125 (Oct.-Dec.) 1951.

Hector presents a series of 11 cases of typical allergy in which he used corticotropin (ACTH) and cortisone. The treatment resulted in complete cure in seven cases, partial improvement in two cases, and moderate improvement in two cases. He employed varying dosages of both cortisone and corticotropin for varying periods. In one case (5.) he had to supplement the use of thyroid extract. He feels that these preparations are important adjuvants to the control of many allergic manifestations. He has not observed any untoward manifestations following the use of either of these preparations.

PERSKY, Philadelphia.

Books

Grundlagen der Strahlentherapie. By Dr. Med. Richard Kurt Kepp, Professor of Obstetrics and Gynecology at the University of Göttingen. Price, $9.00 (38 German marks). Pp. 356, with 134 illustrations. Georg Thieme, (14a) Stuttgart-O, Diemershaldenstrasse 47, Germany.

This book excellently presents a consideration of radiation physics and radiation biology in specific relationship to general radiation therapy. It represents a unified approach to the fundamental principles of radiation therapy which has long been needed.

The first major section of 131 pages is devoted to radiation physics. The remainder of the book considers radiation biology and general radiation therapy.

The portion of the section on radiation physics devoted to the roentgen ray is well done. After a discussion of the nature, production, characteristics, and measurement of the roentgen rays, there is an excellent discussion of their dose distribution in the irradiated subject. The chapter on dose distribution in superficial therapy, medium-voltage therapy, high-voltage therapy, and contact therapy is especially valuable.

The next two sections on radioactive emanations (including natural and artificial radioactivity) and corpuscular rays are not as adequately done. There is no reference to the contributions of Quimby and Failla to radium dosimetry. There are also portions of these sections which have poor readability due to repeated sequential references to the literature. In general, however, the presentation in these sections is adequate.

The remainder of the book, a discussion of radiation biology and general radiation therapy, is a valuable contribution which is well done.

The first chapter discussing general aspects of biologic radiation effects is very contributory.

After a chapter devoted to the effects of radiation upon cells and tissues, there is a section devoted to effects upon specific organs. The portion of this section describing the variation of skin reaction with changes in radiation quality, spatial dose distribution (related to quality, field size, and focal skin distance), and time dose distribution is very worth while.

The concluding chapter (56 pages) discusses the radiation management of malignant tumors. Portions of this chapter also suffer from excessive references to the literature.

A bibliography and an index close the book. The bibliography indicates very meager references to the workers in America.

This book can be highly recommended. An English translation might be in greater demand.

Leitfaden der praktischen Audiometrie. By Prof. Dr. Bernard Langenbeck. Price, 19.50 German marks. Pp. 155, with 77 audiograms and 3 illustrations. Georg Thieme, (14a) Stuttgart-O, Diemershaldenstrasse 47, Germany; Agents for U. S. A., Grune & Stratton, Inc., New York, 1952.

This is a comprehensive manual of practical audiometry. The author has attempted to evaluate the various tests for hearing, but he stresses the use of audiometers in the diagnosis of various forms of deafness. He presents a series of audiograms to illustrate the various hearing defects and their interpretations. This apparently is another addition to the already increasing number of books on this subject. Although it is thorough and the author presents the various theories and tests in a concise fashion, it does not seem to add greatly to our present knowledge on this subject. There is no index but a fair bibliography.

Atlas der Hals-Nasen-Ohrenkrankheiten. Third edition. Edited by Prof. Dr. C. von Eicken and Prof. Dr. A. Schulz van Treeck. Price, 78 German marks. Pp. 200, with 470 illustrations, most of which are in color. Georg Thieme, (14a) Stuttgart-O, Diemershaldenstrasse 47, Germany; Agents for U. S. A., Grune & Stratton, Inc., New York, 1951.

The third edition of this atlas was necessitated by an almost immediate oversubscription of the second edition. It has been carefully reviewed and revised and brought up to date with the newer principles of therapeutics.

This atlas is an extremely comprehensive well-conceived book and is extremely valuable for the student, the general practitioner, and the specialist. Each section is clearly depicted, and the legends are simple, thorough, and easily understood. Each section is divided into two subjects, simple anatomy and normal and pathologic conditions, and both are illustrated in color and with schematic drawings in black and white. Pathologic conditions are described in fair detail, including the etiology, course, therapy, and prognosis. Methods of examination pertaining to the various topics are also included, as is x-ray examination. There is also a page devoted to the new and old nomenclature. The index is extremely comprehensive. The format is clear and easily read. All in all, it is a splendid reference book, and it is well worth while for all practitioners and otolaryngologists.

Directory of Otolaryngological Societies *

INTERNATIONAL

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: México, D. F., Mexico. Time: January, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3, Pa.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: 1953.

Fifth International Congress of Oto-Rhino-Laryngology

Place: Amsterdam, Netherlands. Time: June 8-15, 1953.
President: Prof. Eelco Huizinga.
General Secretary: W. H. Struben, J. J. Viottastraat 1, Amsterdam-Z.
Scientific Secretaries: J. Bijtel and P. G. Gerlings.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Dean Lierle, Iowa City.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James Milton Robb, 315 Lakeland Ave., Grosse Pointe 30, Mich.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 11-17, 1952.

American Board of Otolaryngology

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Roosevelt Hotel, New Orleans. Time: April 21-25, 1953.

American Broncho-Esophagological Association

President: Dr. Fred W. Dixon, 1027 Rose Bldg., Cleveland.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Roosevelt Hotel, New Orleans. Time: April 28-29, 1953.

American Laryngological Association

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 7.
Place: Roosevelt Hotel, New Orleans. Time: April 26-27, 1953.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis E. LeJeune, 49 Audubon Blvd., New Orleans 18.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Roosevelt Hotel, New Orleans. Time: April 28-30, 1953.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Irl H. Blaisdell, 202 Greenwood Pl., Syracuse 10, N. Y.
Place: Hotel Syracuse, Syracuse, N. Y. Time: Jan. 7, 1953.
Southern.—Chairman: Dr. W. W. Wilkerson, Jr., Curtiswood Lane, Nashville 4, Tenn.
Place: Andrew Jackson Hotel, Nashville, Tenn. Time: Jan. 12, 1953.
Middle.—Chairman: Dr. Henry L. Williams, 818 4th St., S. W., Rochester, Minn.
Place: The Drake, Chicago. Time: Jan. 19, 1953.
Western.—Chairman: Dr. Alden H. Miller, 4000 Woking Way, Los Angeles 27.
Place: The Elks Club, Los Angeles. Time: Jan. 24, 1953.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Albert C. Furstenberg, 201 S. Main St., Ann Arbor, Mich.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Roosevelt Hotel, New Orleans. Time: May 1-2, 1953.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Benjamin H. Shuster, 1824 Pine St., Philadelphia.
Secretary: Dr. Louis Joel Feit, 50 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

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Proc. Staff Meet. Mayo Clin. **26**: 361, Sept. 26, 1951.

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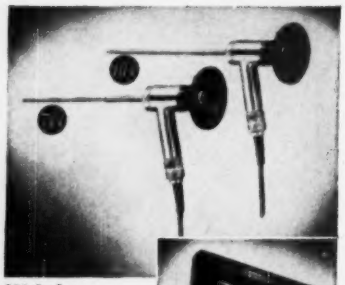

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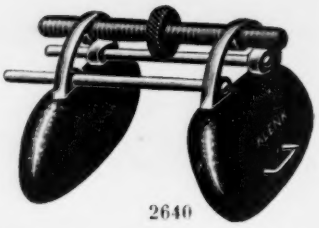

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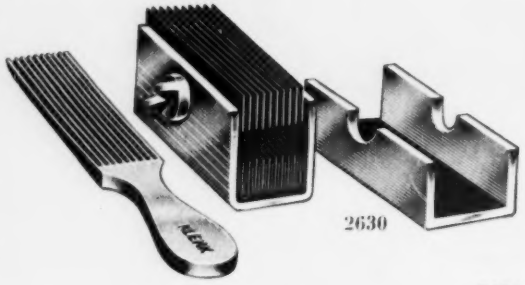

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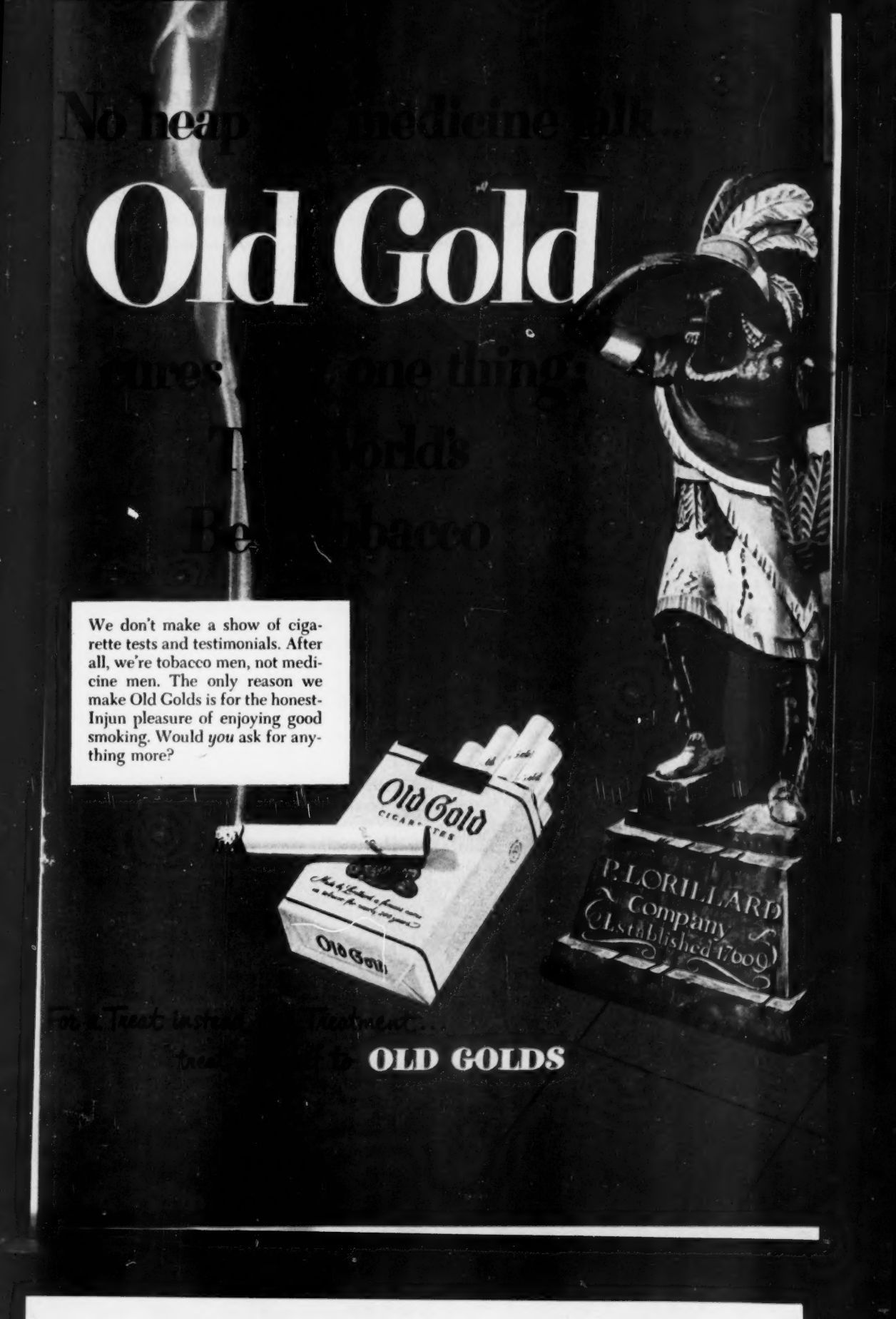
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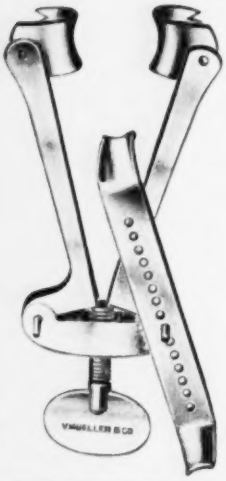

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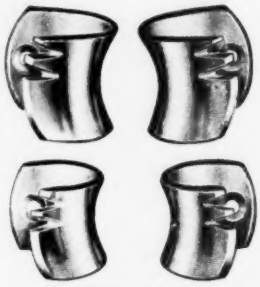

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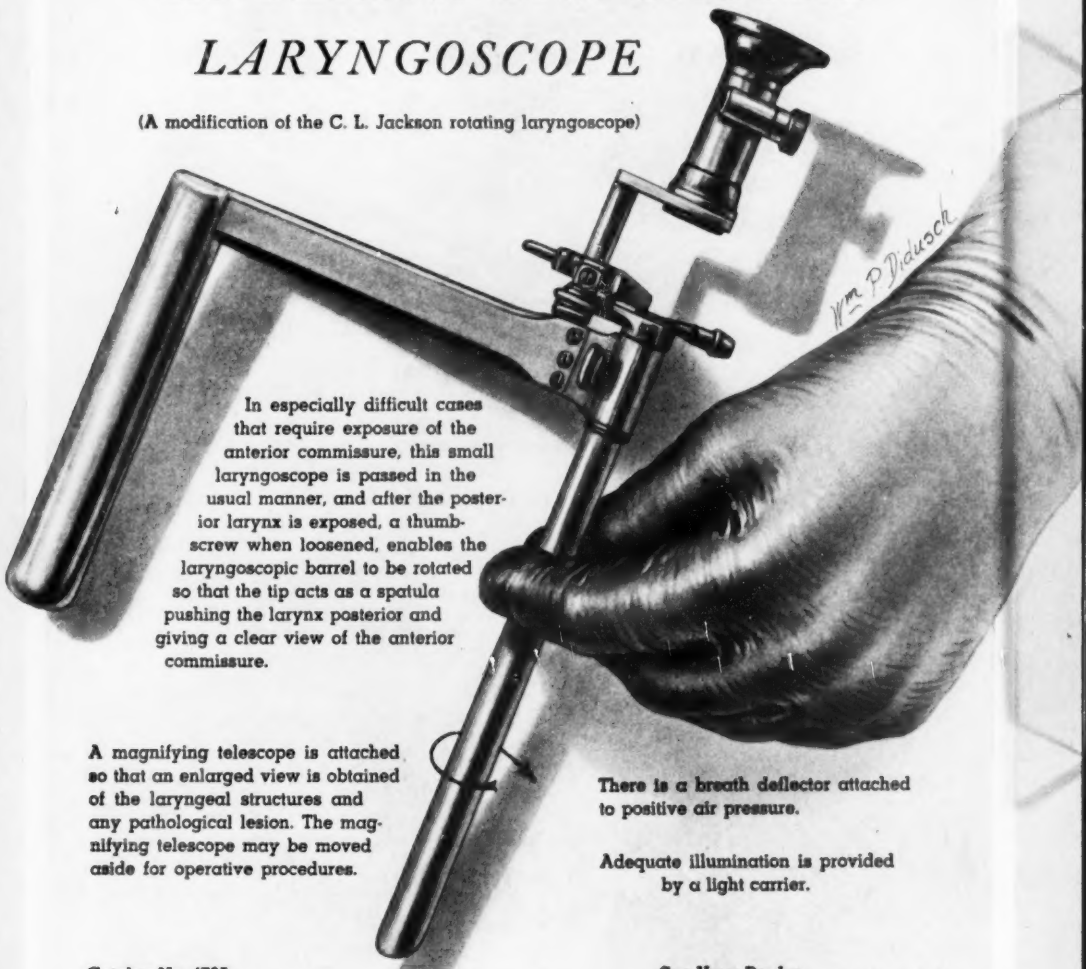

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